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PHONASTHENIA AND ITS TREATMENT BY ELECTROPNEUMOTHERAPY.*

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As physicians particularly interested in the study and treatment of diseases of the ear, nose and throat, we are faced from time to time with conditions of an extremely painful nature, or of a prostrating nature, or of a nature threatening the actual life of the patient, and our literature is filled with treatises on such disease conditions. There are, however, other abnormalities, which, although not actually dangerous from a standpoint of life, may be not only extremely bothersome to the patient but in their consequences may have a tremendous social, occupational and economic effect.

One of the most striking conditions of this type is that of phonasthenia, which I have elected to present to you today for consideration, partly because it has received so little consideration in the English and American literature of our specialty, and partly because of the apparent lack of understanding of its etiology and pathology as well as of its dire consequences.

The treatment of this condition has frequently been highly unsatisfactory. Phonasthenia, called by Flatau, Froeschels and others, functional vocal fatigue, results from the improper use of the voice.

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This implies an improper functioning, not of the vocal cords exclusively, but of any other part of the voice mechanism: respiration, pitch or articulation.

We are prone perhaps to think of phonasthenia as a condition found only among singers or professional public speakers, whereas a much larger group, more apt to be represented in our daily practice and perhaps more difficult to identify, are those who are not dependent upon the voice primarily in their professions or callings, but who unwittingly are daily subjecting the voice to unusual strain. Particular attention is dedicated to this group by Sokolowsky, who calls them the phonasthenics in daily life, as contrasted to the other more limited groups, namely, professional speakers and singers. It is evident then that the condition of phonasthenia may be found not only in opera singers, concert singers, music students, actors, clergymen, attorneys, lecturers, school teachers and auctioneers, but also in salespeople, factory workers, outdoor workers, or in any individual who in any circumstances must strain his voice unusually, such as those who are constantly thrown with a deaf individual or those who must for long periods talk sufficiently loudly to overcome extraneous noises.

As stated above, it is sometimes at the outset difficult to perceive the true state of affairs, but much valuable information can be obtained by a history consisting primarily of several questions, relating not only to occupation, of course, but to the circumstances under which the patient works and lives. In addition to this there are several simple tests.

McMahon has pointed out that when the posterior pillars are observed as the patient says "ah," they assume a position of an inverted U if the voice is properly used, and that if they appear as an inverted V, it is a good indication of improper use of the voice.

The second simple test for a patient whose complaint is suggestive but whose speaking voice offers no clue is to ask him to read aloud or recite, whereupon the voice at once becomes high, loud and hard, the latter referring to the attack, particularly of words beginning with vowels. These are the three cardinal signs according to Sokolowsky: a voice that is too high, too loud, and too hard.

Further observation will show that the patient's breathing is incorrect. Instead of deep and active abdominal breathing, the respirations are apt to be limited to the upper thorax.

The patient himself is rarely conscious of any of these faults, and

his complaint, if a singer, is usually that he tires easily from singing, whereas formerly this was not the case, or that he is conscious of an effort, which was formerly not necessary, or that he breaks on certain notes, or that a tremolo effect to which he was previously not accustomed has appeared when he sings for any length of time. The public speaker also complains of unusual effort and fatigue and of lack of clarity of his voice. The nonprofessional patient of the larger group, such as the stenographer who has a deaf employer, states that during the early part of the day her voice seems normal but in the afternoon or evening she becomes quite hoarse and that her voice sometimes fails entirely. Patients from any of these groups may complain in addition of a scratching, tickling or dryness of the throat, and sometimes even of actual pain in the region of the larynx. As the condition persists and no relief is obtained, psychic elements appear, which at times assume almost unbelievable proportions and may, as one observer has put it, "lead to a complete collapse of the patient's social position," and this factor must be kept constantly in mind.

Most of us have suffered from phonasthenia at some time or other in its milder, transitory form following some voice strain, such as singing in a crowded, smoke-filled room, overenthusiastic cheering at a football game, or even the making of an after-dinner speech if this is done under adverse circumstances, either physical or psychic. When one has strained his voice unduly under any such conditions, he may awaken the following morning finding that with or without pain in the throat or laryngeal region, it is difficult to speak. By this I do not necessarily imply that it is impossible to produce a tone, but the tone produced is not clear and it is produced with considerable effort, which is not normal. When, at this time, we instinctively rest the voice as much as possible, the situation clears up without difficulty, as the acute traumatic inflammation subsides.

With the class of patients, however, under consideration in a discussion on this subject, the matter is usually quite different, because, as has been stated before, they are ordinarily of the class of singers or public speakers; and the auctioneer or salesman, perceiving the same subjective sensations, is often unable to rest his voice; and the opera or concert singer, although actually able to rest her voice, is invariably convinced that this is impossible because she must necessarily appear for the performance in question. I remember, e. g., one of the first patients whom I treated with a marked case of phonasthenia. He was an opera singer brought to our city from Chicago for an open air performance, and his excited mental attitude about

the condition of his voice was expressed in his statement that he was the only man in America who could sing the role in question. This, interestingly enough, is approximately the attitude of most singers, good or bad, even though in this case the gentleman may have been correct in his statement. At any rate, in such a case the vicious circle is at once established: the greater the effort to overcome the condition, the more marked the difficulty, and the more chronic it becomes.

At this point is our opportunity and our duty to recognize the condition and to be able to treat it properly, for with all of the present day statements and statistics about the economic factor of various disease conditions, it is not amiss in passing to call attention to the tremendous economic factor involved when a vocal artist engaged for a single night, with a concert hall sold out to its capacity, finds herself suffering from this unfortunate condition.

In view of what has been said regarding the etiology, it becomes obvious that if the fault lies primarily in the manner of breathing, a correction of this should be made to overcome the phonasthenia. Likewise, slurring and improper articulation can often be corrected by instructing the patient as to the proper use of the lips, the most important factor in proper enunciation. Likewise, correcting habits of talking too loud or too much in noisy surroundings will also help. In singers, the common difficulty is that the individual has been trained or has elected to sing in an improper range, usually one that is too high for the individual, and this error is very widespread.

An infallible test, according to McMahon, is to ask for a deep tone on the sound of "ah." If good resonance is obtained, this voice should be trained in a lower range, and in spite of the fact that the human voice ordinarily has a range at the outer limits of from 64 to 1024 vibrations per second, and in spite of the fact that there has been only one Patti who could sing 1536 vibrations per second, and only one Lukrezia Ajugari who could sing 2048 vibrations, we have all of us seen aspirants. And one thing is certain; namely, that if this persistent use of a pitch that is too high for the individual voice is continued, damage will result.

The best test for a singer is to ask him to sing the scale, as we compare the notes with an organ, a properly tuned piano, or a tuning whistle, whereupon the detoning or distoning is usually noted, most often in the middle register, the difficulty usually arising in the transitional tones and often from the fact that tones which should be chest tones are sung as head tones, and vice versa.

The mistake, of course, has frequently been made by reputable otolaryngologists of attempting to treat phonasthenia as an acute laryngitis. Naturally the treatment is in vain. Inhalations are of no avail, likewise nose drops, sprays, swabs, injections or internal medication, and the reason lies in a correct interpretation of the laryngeal picture gained by a careful examination. In a true phonasthenia, one does not observe an inflammatory condition of the larynx; the vocal cords are no more injected than is the case within the limit of normal individual variation on the basis of occupation, habits, etc. The so-called catarrhal signs sometimes found are not the cause of the phonasthenia and are not the elements to be treated. They are in fact a result of the phonasthenic condition with daily straining, pressing, etc., which causes disturbances of the local blood supply, and a correction of the phonasthenia will correct that just as a correction of the phonasthenia will often cause the disappearance of small singer's nodules which have resulted from the improper use of the voice. The outstanding characteristic feature in phonasthenia is that upon phonation the cords do not meet sharply in the midline. The degree of lack of approximation of the vocal cords, of course, depends upon the individual case, and varies from a flaccid condition to an outspoken, open elliptical space between the cords, or a difference in level of the cords and an over-riding of one arytenoid, and even the most highly trained professional singers seem absolutely incapable of overcoming this. In foreign clinics devoted exclusively to the treatment of voice and speech defects, I have seen many singers from national and municipal opera houses who had suffered from this condition for weeks, supplementing their own muscular efforts to overcome it by sprays, inhalations, and similar measures, all to no avail. All of these measures should be avoided because they do not help the actual condition, and therefore add to the discouragement and psychic depression of the patient. Likewise, operative procedures for anything other than the actual improvement of resonance by the removal of an extreme deviation of the septum or hypertrophied posterior tips should also be avoided. If the correction of breathing, articulation and pitch are not sufficient, and we are dealing with an obstinate, chronic functional fatigue or phonasthenia, compensation measures alone will suffice to overcome it.

And yet, when one considers the physiology of such a condition, its treatment becomes apparent and seems quite simple. The story is this: the singer continues with a strenuous rehearsal or performance in the presence of a mild upper respiratory infection, which responds quickly within the next few days to treatment. All inflam-

matory signs have disappeared but the vocal difficulty persists, and examination reveals the picture described above. What has happened is that the vocal cords, under strain, have become somewhat edematous, and as long as the slight edema remains, the vocal cords meet in the midline and comparatively adequate tones can be produced. When the edema subsides, however, the space which it previously occupied is not filled upon phonation or, in other words, the cords do not approximate completely, and difficulty ensues. No amount of forcing on the part of the patient can overcome this ordinarily, and there ensues an additional psychic element.

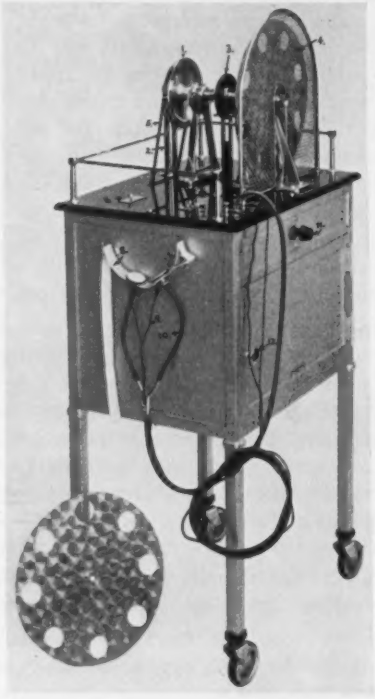
I like to think of this as comparable to the situation present when a fractured arm has been immobile for weeks in a cast and the cast is removed. The fractured bone is healed, the muscles are present and the nerves are present, but normal physiological function is lacking and, in order to restore it, compensation treatment is given in the form of electrical stimulation to the nerves and muscles, and massage or light treatments are utilized to increase the local blood supply. Exactly the same fundamental principles of therapy should be applied to the larynx in cases of persistent phonasthenia.

This is not, of course, a new contribution, for it has long since been recognized, particularly by the European workers, that Faradic current stimulation to the region of the thyroid cartilage, combined with measures designed to increase the local blood supply, is the method of choice to treat and overcome obstinate phonasthenic conditions. They also recognize, however, that to be effective the Faradic impulse must be introduced while the vocal cords are vibrating and at a certain phase in their vibration, and this concept I take to be the important difference between the results obtained by those men who have followed it and those who have merely "given electric treatments" without regard to the physiology of phonation.

It was just this fundamental factor in the correct treatment of phonasthenia that instigated the designing of the pneumotherapy apparatus which I am presenting to you today, for I felt that it would be of distinct advantage to the patients and also to the members of the medical profession treating them if a means could be devised to assure the fact that the Faradic impulses would indeed be introduced at the proper phase of vibration of the vocal cords.

The apparatus itself is comparatively simple. There is, as you see, a motor-driven shaft which carries on the anterior end a large aluminum disk which is perforated and which revolves as the shaft turns.

In close proximity to this is a second stationary disk of the same size and character. Next on the motor shaft is mounted a disk made of a nonconductive material in which have been set eight small metal plates. In contact with this disk are two poles, the first of which



1. Balance wheel. 2. Air pump. 3. Disk with contact plates. 4. Aluminum siren disks with wire mesh guard. 5. Driving belt from motor. 6. Motor switch. 7. Faradic current switch. 8. Neck band carrying electrodes and vacuum cups. 9. Faradic current lead. 10. Air hose from pump. 11. Rheostat knob controlling motor speed. 12. Door to cabinet enclosing the motor and Faradic current apparatus.

carries the Faradic current to the contact disk, the other carrying the current from this, through the wires, to two metal electrodes, mounted on an adjustable neckband which makes it possible to place them at the sides of the thyroid cartilage. At the far end of the motor shaft is an air pump, which, by means of its positive and

negative phase, supplies air in an interrupted stream through tubing to vacuum cups, incorporated within the electrodes mounted on the neckband.

When the patient is seated before the apparatus and the neckband is properly adjusted, the motor switch is turned on. As the large aluminum disk revolves in close proximity to the stationary disk, it acts as a siren, and a note is produced, this note depending, of course, for its pitch on the speed of revolution, which is controlled by a small rheostat mounted on the front of the apparatus. At the same time the air pump is in operation and an air massage is effected at the desired point. The patient now hums the note produced by the siren, if he is sufficiently trained to do this. If not, by means of the rheostat, the speed of revolution is so regulated as to adjust the note produced by the siren to that which the patient hums. And, finally, if the patient is unable to sing, he may talk, recite, or count in a monotone, and again the rheostat regulation makes possible the adjustment of the siren note to the tone of his voice.

When this has been done, the Faradic current switch is thrown, and inasmuch as the electric impulse is released at the electrodes at the sides of the neck only at the instant when the metal plates set in the revolving disk are in contact with the afferent and efferent poles, and inasmuch as these plates have been carefully spaced with a micrometer and synchronized with the eight perforations in the larger aluminum disk, there is mechanical and physiological assurance that the impulses are coming to the larynx always in the same phase of vibration of the vocal cords. The stationary aluminum disk containing eight perforations is interchangeable with one containing sixteen, thus providing for a lower and higher range of notes.

The motor which drives the shaft by means of a belt is within the cabinet, as is also the small, dry-cell Faradic current apparatus, the strength of the Faradic current being adjusted in the usual manner by means of the small coil rheostat. The guard placed before the aluminum disks is obviously for safety purposes, to protect a patient with inquisitive fingers. The entire apparatus is mounted on four wheels to facilitate its transportation about an office or clinic.

The apparatus is manufactured according to my design by the Max Wocher & Son Company in Cincinnati.

A single treatment consumes from ten to fifteen minutes, and does not require the presenec of the physician or an attendant during this time. Often one treatment suffices, sometimes several on successive or alternate days are indicated, but it is interesting to

observe how readily and quickly the detoning and lack of clarity of the notes is overcome during a single treatment. It is also interesting to hear the voluntary comment of nearly every patient who arises from the treatment chair on the comfortable, relaxed sensation and disappearance of the previous sense of irritation of the throat and larynx. In fact, an actual area of erythema surrounding the vacuum cups is usually demonstrable.

The apparatus has been in use in our office for fifteen months, and on the basis of the beneficial results obtained during that time, it is offered as a standardized method of treating this bothersome and very important condition in lieu of the haphazard methods which have unfortunately been necessary frequently in the past.

In closing, I wish to present briefly several case reports of patients who have been treated with the electropneumotherapy apparatus and who represent different aspects as far as occupation and etiology are concerned.

Case 1: Mrs. E. B. D., age 40 years, soloist in church choir. Had grippe one week before examination; complained of persistent hoarseness and of effort required in speaking; had been unable to sing. Examination showed no signs of acute inflammation; there was faulty approximation of the vocal cords. A ten-minute treatment with electropneumotherapy apparatus was given. Patient commented on relaxed feeling of throat and next day reported that her throat had felt more comfortable and her voice had been better than it had been for six months. She added that all of her life the larynx had been her weak point, and that any mild infection produced the symptoms described above.

Case 2: Miss J. H., age 22 years, senior student at college of music. For several weeks patient had been rehearsing strenuously for an operetta to be given three days after this visit. She complained of fatigue after singing for a short time and unusual effort required, together with lack of clarity of tones. When tested with the tuning whistle she was found to be detoning on every note in the middle register. Ten-minute treatment with electropneumotherapy apparatus was instituted; no other measures were used. At the end of the treatment she was again tested with the tuning whistle, and every note in the middle register was found to be true. Two subsequent treatments were given with complete relief from symptoms.

Case 3: Miss G. W., age 37 years, piano accompanist. Complained of persistent irritating, nonproductive cough, which had been treated

for two weeks with various medications without effect. Fifteen-minute treatment with the electropneumotherapy apparatus resulted in the report on the following day that the cough was very much decreased. Treatment repeated with complete cessation of cough.

Case 4: Miss E. D., age 31 years, school supervisor of music. Complained of vocal fatigue, foreign body sensation in throat, and hoarseness following upper respiratory infection, symptoms persisting in absence of acute inflammation. She was treated with the electropneumotherapy apparatus with marked improvement. She had two subsequent treatments with relief of symptoms. The same condition and same result followed two subsequent head infections.

Case 5: Mr. R. H., age 46 years. Complained of hoarseness of three weeks' duration, during which time he was treated by family physician, and had grown progressively worse. Examination showed swelling of the right arytenoid and ventricular band with several ulcerations in the interarytenoid space and marked inflammation of the surrounding area. The diagnosis of tuberculosis was substantiated by bacteriological examination and chest examination, together with X-rays, which showed a chronic bilateral pulmonary tuberculosis. Under general treatment, healing of the primary and secondary lesions took place; the larynx was free of ulceration and proliferation, and the cords were slightly thickened and approximated incompletely. A series of treatments with the electropneumotherapy apparatus corrected the faulty approximation with subsequent improvement in the voice.

Case 6: Mrs. A. B., age 38 years, concert singer. Complained of effort required in singing and extreme hoarseness. Examination showed the larynx normal in color and cords normal except for incomplete approximation upon phonation. Treatment with electropneumotherapy apparatus for ten minutes caused improvement in voice. The following day the patient reported marked improvement; the treatment was repeated. On the next day the cords were found to be approximating perfectly; the third treatment was followed by complete return to normal function.

SUMMARY.

Phonasthenia, or functional fatigue of the voice, is a condition which is not only extremely disagreeable subjectively to the patient, but one which may be attended by serious social and economic consequences. It is not exclusively an occupational disease in the sense

of being confined to singers and professional speakers, but also frequently exists and is frequently overlooked in individuals in many walks of life, who daily unconsciously strain the voice or use it improperly.

When the etiology and pathology of phonasthenia is understood, it will be apparent that the correction of improper respiration, articulation and pitch will frequently relieve it. When it is persistent or chronic in character, compensation treatment by means of the Faradic current, together with measures to increase the local blood supply is indicated, and a new electropneumotherapy apparatus is presented herewith which makes possible the correct and synchronized electric stimulation to the larynx.

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PLASTIC RECONSTRUCTION OF THE EXTERNAL AUDITORY MEATUS.

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Injuries about the head and face since the advent of automobiles have become much more common in civil practice; consequently, new problems in plastic reconstruction have presented themselves. The case which I wish to report is one in which the entire cartilaginous meatus was destroyed in an automobile accident.

Heretofore, traumatic loss of the external auditory meatus has not been a very common occurrence. In fact, I was unable to find in the literature any report of a plastic reconstruction of the canal. This case, therefore, in which the external auditory meatus was reconstructed by the use of a pedicle skin graft, seems worthy of description, especially since the method is so simple and yet successful.

The loss of the external auditory meatus was caused by an automobile accident in which a rather wide piece of broken glass entered the tissues immediately in front of the ear, dissecting the ear, together with a portion of the scalp posterior to it free from the skull and entirely destroying the cartilage and skin of the canal. The injury was taken care of at the time by a general surgeon who secured a good coaptation of the parts. However, on account of the contused nature of the wound and the entire cartilaginous portion of the meatus being destroyed, in order to maintain a patent opening it was necessary to place a piece of rubber tubing in the tissue to take place of the destroyed external auditory meatus. The patient first consulted me seven weeks after the accident because the insertion and wearing of the tube was becoming painful. The healing of the tissues was gradually closing off the opening and clamping down on the rubber tube.

At this time the face and scalp wounds were completely healed. Examination of the external ear showed the tragus and a portion of the antitragus missing. The cavum conchae was destroyed and the cartilaginous external auditory meatus was represented by a circular

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opening 4 mm. in diameter surrounded by scar tissue. The appearance of the ear at this time is shown by the drawing in Fig. 2, which shows also the line of the wound. There was a 4 mm. rubber tube in the meatus and from it came a small amount of purulent discharge, caused by the irritation of the rubber tube to the newly-formed scar tissue. The bony external auditory meatus and the tympanic membrane appeared to be normal.

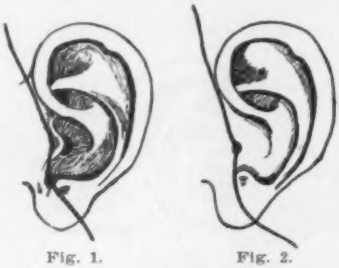

Fig. 1. Fig. 2.

Fig. 1. Normal ear showing the line of injury.
Fig. 2. Showing appearance of ear at time of first visit. Note the small sized opening and loss of tragus.

Upon removal of the tube for twenty-four hours, the opening rapidly closed and a forceful dilatation was necessary in order to reinsert the tube. While closure of the opening could not be permitted, continuous wearing of the tube was undesirable because of the discomfort it produced. Therefore, it seemed advisable to devise some method for the reconstruction and epitheliazation of the opening which represented the external auditory meatus and the following operation was performed with gratifying results.

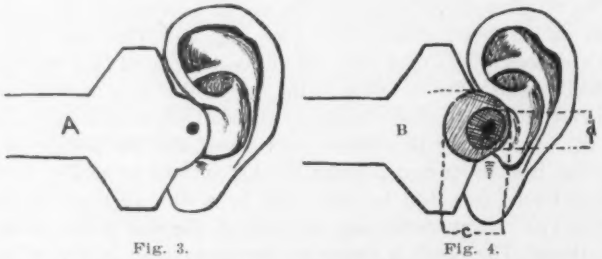

Fig. 3. Fig. 4.

Fig. 3. Showing pattern of flap in place for incisions.
Fig. 4. Showing ear after enlargement of meatus.

After experimenting for some time with various shapes of flaps, the ones pictured here as A and B were finally devised and they worked admirably. Prior to the operation these two patterns, A and B were cut from lead foil about one-eighth-inch thick or less and sterilized with the instruments. A 0.5 per cent novocaine infiltration anesthesia was used for the operation. Pattern A was first superimposed over the ear and adjacent skin as shown in Fig. 3 and the incisions for the flap outlined. Pattern B (see Fig. 4) was then placed in position, a circle of scar tissue around the meatus

Fig. 5. Fig. 6.

Fig. 5. Showing the flap dissected free.

Fig. 6. Showing the flap made into tube (or canal) prior to being placed into newly formed canal. The flap is stitched together at top and bottom with No. 00 plain catgut, the knots placed on the skin surface of the flap.

removed superficially (see area c, Fig. 4) and the meatus considerably enlarged by the removal of scar tissue to a diameter of about 15 mm. and down to the beginning of the bony canal (see area d, Fig. 4). A full thickness skin flap corresponding in shape to Pattern B was then dissected up as in Fig. 5. An epithelial tube, skin surface to the interior, was formed from this flap by approximating the corners x to x' and y to y', holding them in position by a suture of No. 00 plain catgut placed in the top and bottom of the tube, the knots on the inner (skin) side (see Fig. 6). This tube with its attached pedicle was then slid posteriorly and placed into the newly-formed meatus. The mobility of the skin in this area making only a moderate amount of undermining of the skin edges necessary to permit this easily. The skin defect was closed and the pedicle of the skin flap held in its new position by skin sutures as in Fig. 7. The epithelial tube was held in place only by a firm pack of iodoform gauze. The convalescence was uneventful, the skin graft taking in its entirety. The result is shown by the photograph in Fig. 8, taken eleven months after the operation.

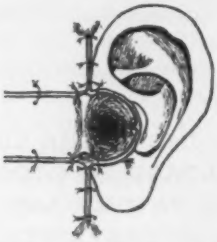

Fig. 7. Showing flap sutured in place.

The photograph shows a slight tendency for the meatus to collapse, which is due in part to the absence of cartilage and in part to the tension of the flap in healing, exerting a pull anteriorly. An excessive amount of collapse can be avoided by the removal of sufficient tissue posterior to the meatus (see area c, Fig. 4) and placing

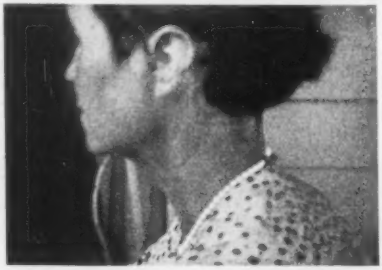

Fig. 8. Photograph taken eleven months after the operation.

the hole in Pattern A not directly over the meatus but offsetting it posteriorly about 5 mm. Care must also be exercised in enlarging the meatus sufficiently before the epithelial tube is slid into place in order to avoid a possible stricture formation at the junction of the new with the bony meatus.

A DISCUSSION OF SOME OF THE RECENT DEVELOPMENTS IN REGARD TO OTOSCLEROSIS, CHOLESTEATOMA AND THE STATIC LABYRINTH.*

Dr. George M. Coates, Philadelphia.

The Chairman of the Executive Committee of the Section on Otolaryngology of the College of Physicians of Philadelphia suggested to the writer the above title for the joint meeting with the Section on Otology and Laryngology of the New York Academy of Medicine in the hope that this modest essay might afford a varied field for discussion that would prove of interest to that audience. As the title suggests, it is not a report of original work but an attempted resumé of certain phases of the work of several distinguished investigators; work that should be of interest to, and that every otologist should take cognizance of. With this brief explanation and apology, I shall proceed with the three assigned subjects.

OTOSCLEROSIS.

This chapter is based upon the work of Guild[2] and Polvogt[1] in Baltimore. The usual picture of the pathology of otosclerosis accepted by otologists and upon which conception the diagnosis of this disease is based, is that the pathology takes place and is confined to the area surrounding the oval window, eventually causing ossification of the annular ligament with fixation of the footplate of the stapes in the oval window, thus interfering with air conduction of sound, giving a negative Rinné test, with prolonged bone conduction and a positive Gellè test. It is recognized, of course, that other middle ear pathology, such as firm fibrous adhesions around the stapes, may simulate the findings that are supposed to signify otosclerosis.

Polvogt and Guild have had the opportunity of studying, by serial section, many temporal bones of patients of whom careful audio-

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metric records have been made and in some of these have discovered definite areas of otosclerosis that did not cause the usually accepted symptoms or were not found in the usual locations. Guild has reported eight cases, to which Polvogt has added two more, where the history, familial and personal, as well as clinical symptoms, did not suggest otosclerosis, but where definite otosclerotic areas were found on sectioning, some of these being at a very early stage, some active and others entirely quiescent, although evidently of long duration, at least partially confirming the well known clinical fact that the disease is at times self-limited, or that it is not uniformly progressive.

Polvogt reported two such cases at the Academy meeting. One was a child, age 8½ years, where tests showed hearing within normal limits. This patient died of a glioma, but definite otosclerotic areas were found around the oval and round windows, although the footplate of the stapes was not involved, and there were no signs or symptoms by which a diagnosis could have possibly been made. This was, of course, a very early case, which might have progressed later in life to the point where it was diagnosable. The second case was 45 years old when tested, had normal limit hearing, no familial history of deafness, no tinnitus. Here an area of otosclerosis was found, involving the fissura ante fenestrum but not involving the stapediovestibular articulation. This case may have been of long standing; we have no means of knowing this; but it is at least evident that unless, or until, the oval window, the footplate of the stapes or the round window, is involved, our usually accepted diagnostic signs fail us completely. In fact, the practicing otologist does not see such cases, for they are not deaf, unless from other causes.

In one of Guild's cases, with normal hearing, areas of otosclerosis appeared to be in a stage of very active growth. Overlying these, the mucosa of the middle ear was hyperemic. Wittmaack believed that venus stasus was a factor in the causation of otosclerosis, and in this case there was greatly increased intracranial pressure from a brain tumor which might easily have caused such stasus. In another case, that of a man, age 68 years, the disease had invaded the margin of the oval window on each side, causing at least some ankylosis, without appreciable loss of hearing for conversation. There was no evidence of recent activity anywhere and Guild concludes that the process may have been quiescent for even several decades, although he cannot venture any opinion as to why this should be.

These cases are all classed as "early cases" because of the lack of clinical symptoms, although they may have been old pathologically.

These changes may begin as early as the first year of life, and they do not always first appear near the anterior margin of the oval window, nor is the fissura ante fenestrum always involved.

So much for the development and pathological findings in early otosclerosis about the oval window, which, when it reaches the stage of ankylosis, we can usually diagnose in uncomplicated cases. What of otosclerotic areas in other regions affecting the cochlea and producing nerve type deafness? The reports of Guild and Polvogt do not mention these, their studies for the present being confined to the region of the fenestrum ovalis; but Nager, of Zurich, when in this country some years ago, showed us in this hall some beautiful sections of the cochlea showing otosclerosis, which, when present, caused nerve deafness that could not be distinguished clinically from that produced by any other cause.

CHOLESTEATOMA.

In his recent paper before the Academy, Almour, basing his work on Wittmaack's theories of pneumatization of the temporal bone and some practical applications worked out in Kopetzky's clinic, advocates the utilization of the cholesteatomatous matrix for the epidermatization of the radical mastoid cavity. This opens up various topics for discussion, only one or two of which may be commented upon at this time. The normally pneumatized mastoid process is formed by the conversion of the embryonal myxomatous connective tissue under the epithelium, which fills the middle ear and antrum at birth, to the adult type found later on in the middle ear and mastoid cells. During the process this embryonal tissue invades and replaces the bone marrow in the small, undeveloped infantile mastoid process, contracting as it develops and being drawn into the intratrabecular spaces and eventually lining them. From these the mastoid cells grow, all, of course, communicating directly or through each other, with the antrum and middle ear. Any interference with this normal development produces either a diploic or sclerotic mastoid; in other words, an undeveloped mastoid process, or possibly a mixed type, depending upon the stage of development when the infection occurred. An uninfected foreign body such as meconium or vomitus entering the middle ear at birth, will cause an interference with the second stage of development so that the fibrous tissue which has replaced the fatty bone marrow will persist in the intratrabecular spaces, be subject to osteoplastic activity of the growing *osseous trabeculae* and result in a sclerotic mastoid process.

If this accident happens after the development of some pneumatic cells, the process will be halted at this point and a mixed type result. When a microbic infection takes place before development has taken place, resulting in an otitis media, healing induces increased fibrosis, which interferes with the ingrowth of the subepithelial tissue into the mastoid process, resulting in the retention of the infantile or diploic type of mastoid. It is thus believed that a sclerotic type mastoid process is the result of an early acute infection that interferes with pneumatization, rather than of a chronic mastoid infection that produces a sclerosis by obliterating pneumatic spaces already formed. Thus a sclerotic mastoid process is not viewed as pathological in itself or the result of the chronic infection in the middle ear or antrum.

Basing their radical mastoid technique upon this theory, or these facts, if they are accepted as such, Kopetzky and Almour, where it can be proved by X-ray that the mastoid process is indeed nonpneumatic and wholly sclerotic, confine their radical mastoid operation to the removal of the thick cortex overlying the antrum, without any attempt to excavate the remainder of the process which is considered healthy bone, not calling for removal. The cholesteatomatous mass is removed with care from the uncovered antrum, the posterior canal wall reduced and the middle ear cavity treated in the usual orthodox manner. This procedure materially shortens the time of operation and leaves a much smaller area to be epidermatized, which should also shorten the time of convalescence. It is considered very necessary that the plastic flap be made with skill and care, and that the meatal opening be ample in order to provide adequate escape for epithelial debris, as otherwise cholesteatoma may redevelop. This failure of a proper avenue of escape is the original cause of the production of cholesteatoma, which results from an ingrowth of epidermis through a marginal tympanic perforation in an effort of Nature to line the cavity and cause cure of the infection. In other words, it is an attempt at a reparative process, which may succeed perfectly if the avenue of escape for exfoliated epithelial debris is adequate. Following this line of thought, these observers advocate the retention of the cholesteatomatous matrix, which is nothing more than an epidermal covering similar to the one which the operator hopes will eventually line his radical cavity to produce a dry ear. Why destroy a perfectly good lining membrane already present and then try to produce another, which will, at best, be but equally good Why, indeed?

The old teaching, following the observation of Kirchner,[4] was

that the matrix must be removed in toto, together with the underlying bone because this matrix reached down into the intratrabecular spaces and, unless removed, would cause a recurrence of the disease. We now believe that this is unnecessary, the cause of recurrence of the cholesteatomatous mass being an inadequate outlet.

This subject is well and concisely presented, but is not all new, although never, as far as I know, have all these points, briefly sketched here, been assembled into one logical whole. For a long time it has seemed to me, and to others, that the point made above in regard to the sclerotic mastoid was correct, although the technique for meeting this condition developed in somewhat different directions. Tobey combines ossiculectomy with an intratympanic antrotomy to remove middle ear pathology and provide the free drainage desired, and he advocates leaving the matrix in situ. I have also consistently advocated this plan of attack when sure that the pathology was entirely confined to the tympanic cavity and antrum. It is often most efficacious. Babbitt, in selected cases, is still more conservative with his attic drainage procedure, endeavoring thereby to remove obstructing pathology and provide drainage with the least destruction possible.

Where a radical operation seems indicated, I have taught for a long time that the sclerotic process should not be excavated, if it contains no cell, but that the operation should proceed along the lines outlined above.

I was a convert to the idea of leaving the matrix in situ, when I first became acquainted with it from the report of Dundas Grant,[3] who advocated its retention as an epithelial lining in 1920. This was only to be done where the matrix, after the careful removal of the cholesteatomatous mass, was dry, pearly gray and glistening. When thus employed in place of a skin graft, recovery was very rapid and the resulting cavity was smoother, dryer and whiter than usually occurs. Dundas Grant remarks that this favorable formation is only found in cases of long standing. These results have been confirmed by J. F. O'Malley and D. R. Patterson. Grant's original case was done about 1905. He does not advocate it for all cases, but only where there is a good matrix, which is adherent.

I have used this method on occasions in past years but was not strikingly successful with it and allowed it to fall into disuse. Credit is due to these workers for presenting it to us in such convincing form.

STUDIES OF THE EQUILIBRIAL MECHANISM OF THE FROG.

McNally[5] and Tait have reached definite conclusions upon the function of the utricle (at least in the frog), and upon some other portions of the static labyrinth. In the first place, these experiments seem to show that the saccule (with its macule and otoliths) has no function in the production of equilibration, but that it appears to be a rudimentary part of the auditory system — or cochlea — in fishes and other lower organisms.

Response to angular acceleration in rotation (slow speed) is a function of the semicircular canals, whereas response to centrifugal force (rapid turning) is otolith stimulation in the utricle. The response to gravity gained by tilting the table is similar to the above, as it is abolished by cutting the nerve to the utricle.

As we have long known,[6] sensory hair cells, similar in general structure to those found in the cristae of the *ampullary sacs,* are found in the utriculus and sacculus. Each collection of hair cells, together with the supporting cells, is designated as a macula, the macula utriculi being found in the utriculus and the macula sacculi being found in the sacculus. Lying among the hairs of the hair cells are found masses of small crystals of calcium carbonate, the otoliths or otoconia. The utricle and saccule in man have two otoliths, but in animals there are frequently more otoliths.

The static function arises from the semicircular canals, while the otoliths serve as a kinetic mechanism. The usual conception of utricular function is that it is concerned with posture. These organs are assumed to give us information regarding the position of the head when at rest and when making progressive — that is, non-rotatory — movements, supplementing, therefore, the functions of the semicircular canals on the supposition that these latter act especially in movements of rotation. According to this view, the otoliths act as a means of mechanical stimulation of the hairs. Being heavier than the endolymph, they press upon the hairs with a force varying with the position of the head and thus give rise to sensations adapted to the maintenance of equilibrium. A sort of continuous discharge of such reflexes has been assumed to occur from the contact of the otoliths with the hair cells in the dynamic organ of equilibration.

If both labyrinths are removed from a frog, it may assume normal and unusual positions but it maintains its position in spite of the lack of labyrinths and therefore the position is due to a neuromuscular mechanism. Change is the essence of stimulation. If the

utricle is responding to a steady pressure, then it is the only known sensory epithelium that is so acting.

When a normal frog is placed upon a plane and the plane is tipped slowly so that the movement is below the threshold of stimulation of the canals, the frog responds by discontinuous shifts. If the utricle were steadily stimulated the shift would be steady and not discontinuous.

If we now remove one labyrinth from a frog, the animal leans to one side. By manipulating such a frog and bringing its posture to the midline the frog maintains its new symmetrical position when released. The moment the head is jarred or moved, however, the frog regains the asymmetrical position. A decerebrate frog is normal in labyrinthine movements but voluntary movements are curtailed. It is the necessary jar of the utricular otolith that causes response. There is no steady pressure but there is a sudden stimulus.

By removing all six canals in a frog, only the utricules remain as active vestibular end organs. The posture is normal at rest but any spontaneous movement causes the animal to sway and the jumps are not straight. The canal response is so rapid that response occurs in 1/25,000 of a second.

In the frog, maintenance of position is evidently a muscular affair. When the canals are present, they act to keep the head level and prevent the sway which the utricles would otherwise bring about. The static mechanism is evidently muscular.

McNally has summarized his ideas upon the utricle as follows:

1. It has been shown that a delabyrinthized animal (utricle and saccule removed) will maintain good posture. This posture is maintained by muscular effect.

2. How is the utricle stimulated? When a frog is stimulated it responds with a shift. The response is discontinuous. The presumption is a shift of otolith on the macula (observed when slowly tilting the frog).

3. Stimulation causes assumption of new posture. This new posture is dictated by the utricle, but maintained by the muscles.

4. The utricle does not respond to constant stimulus. It is also observed that no other epithelium responds to a constant stimulus.

5. If the labyrinth on one side is removed, the animal will lean to the same side. This can be corrected by grasping the head and

bringing it back to symmetry. It will then maintain that position for some time. However, if the table be jarred or a spontaneous movement of the head be made, the utricle will be stimulated, causing change of posture to the same side.

6. If all the six canals be removed, with any spontaneous movement there will be a marked swaying of the head. If the utricle is removed the swaying disappears. This is due to the fact that any movement of the head out of the horizontal plane causes a marked stimulation of the utricles, showing that the utricle responds to movement.

The utricle responds to movement and because it changes, rather than maintains, posture, it is kinetic and not static.

McNally further asks and answers the following five questions:

1. Does the utricle maintain posture? No; the muscles do.

2. How is it stimulated? By movement.

3. Is it constantly signaling? No!

4. What is its effect on the organism? It breaks up existing posture and dictates the new one—a more suitable one.

5. Is it really a static organ? No; it is a kinetic organ.

I am unable to comment upon or criticize this experimental work of McNally and Tait, but it all seems very logical and clarifying in our ideas of labyrinthine function, and while there is a great deal yet to be discovered in this field, I have the greatest admiration for this work and believe that it has added greatly to our store of knowledge.

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INTRACAROTID TREATMENT OF MENINGITIS AND CHANGES NOTED IN THE CAROTIDS FOLLOWING INTRACAROTID THERAPY. MEASURES TO PREVENT THESE CHANGES.*†

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It is a distressing fact that purulent meningitis at best is difficult to treat and the prognosis is most unfavorable. The mortality from this almost hopeless condition is terrifically high, due to anatomical and mechanical factors which cannot easily be overcome. Various methods have been employed in the treatment of meningitis, those current being sera, vaccine, bacteriophage, spinal drainage, cisternal puncture, restriction of fluids, Kubie's[1] method of forced drainage of the central nervous system, frequent intravenous medications and intracarotid sero- and chemotherapy.

There is a reason to believe that intracarotid therapy offers a ray of hope, with a faint chance that recovery may occur.[2]

Dr. Kolmer[3] believes, "that while the intracarotid route of medication has not solved the treatment of septic meningitis, yet it has proved a safe and clinically applicable procedure offering some hope and advantage over ordinary forms of treatment."

We agree with Dr. Kolmer that intracarotid injections are safe. They are feasible and can be used indefinitely as the carotids can stand a reasonable amount of abuse without injury to the artery or untoward signs systemically. However, there is a sporadic case here and there where the artery becomes injured as a result of repeated manipulations.

The question has time and again been asked, what happens to the carotid after exposure, repeated manipulations and frequent intracarotid injections? In answer to this, I should like to quote a case where thrombosis developed in one of the carotids as a result of treatment.

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*Read before the Philadelphia Laryngological Society, Jan. 3, 1933.
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Patient R. C., female, white, age 19 years, was admitted to the Mt. Sinai Hospital, Nov. 28, 1931. Three weeks previously the right ear drum had been incised, since which time it had been draining purulent material. On day of admission, patient vomited three times and the temperature rose to 103° F. When she entered the hospital she was markedly toxic; she was clear mentally but displayed rigidity of the neck, positive Kernig and Brudzinski signs, absent knee jerks, and marked skeletal muscular hypotonia with slight left-sided facial weakness. The right ear was violently inflamed; there was an acute mastoiditis with an acute frontal sinusitis. The leukocyte count was 21,400, with 92 per cent polymorphonuclears. Lumber puncture revealed the spinal fluid cloudy, under increased pressure, with a count of 1,600 white blood cells, nearly all polymorphonuclears. Direct smear was positive for Gram positive cocci in chains. Immediate right mastoidectomy was performed with exposure of the dura and lateral sinus. A forty-eight-hour culture of the original spinal fluid was taken before operation and was returned densely positive for hemolytic streptococci. The general condition of the patient became worse. She was now stuporous and the meningeal signs were more advanced. Because of the hopelessness of the case and the almost certain fatality, once a diagnosis of streptococcic meningitis is made, the senior author exposed both common carotid arteries under local anesthesia in order to administer medication directly into the circulation of the brain and meninges.

The program of treatment instituted consisted of intracarotid injections of 10 cc. each of 0.5 per cent solution of acriflavine base and colloidal solution of iodine twice daily, using each artery alternately every twelve hours. Blood transfusions and intramuscular injections of antistreptococcic serum were given every day. The night after two intracarotid injections were given, the patient's general condition seemed to improve. However, two days after exposure of the carotid arteries, the patient exhibited a palsy of the sixth nerve on the right side with a left pupillary dilatation. Five days after the operation there was complete recovery of the right sixth nerve with a partial paresis of the left sixth nerve. The meningeal symptoms gradually cleared; only a slight rigidity of the neck remained. The interesting feature worthy of note was that on the fourth day after operation we encountered great difficulty in injecting the right common carotid artery. This was about the time that the sixth nerve palsy, partial facial palsy and paralysis of the left twelfth nerve occurred. From these facts it may be speculated that

the nerve palsies may have been due to ischemia of the right side of the brain, caused by a thrombus formation on the intima of the right common carotid artery induced by the injections.

Following this case, which made a recovery despite the thrombosis, we made a mental reservation that if we should happen to treat by this method other cases who would not recover, we should make every effort to obtain the carotid vessels for histologic study of the changes. These thoughts were timely, because unfortunately soon after on our service at Temple University Hospital, we were confronted with a series of three fatal cases of otitic meningitis treated by carotid therapy; we are presenting a study of these vessels and, in addition, we also examined the carotid arteries of several dogs where injection therapy was carried out.

The following histopathological observations were made as a result of the studies:

a. There was an intense periarteritis invading the outer coat of the vessel wall with a tendency towards necrosis of the tissues.

b. An atheroma with a weakening and softening of the vessel walls with a tendency to aneurysm. (Dr. Kolmer[4] reports an interesting and unusual complication on a patient who developed an aneurysm of the left common carotid artery, doubtless the result of the large number of injections.)

c. There was an invasion of the blood elements within the vessel wall with secondary fibrosis without changes in the intima.

In another specimen we found that the external inflammatory elements were lacking but there was a striking denudation of the intima and the tendency to well organized clot formation on the internal vessel wall with thrombosis.

The following is a resumé of the three cases whose carotids we obtained on postmortem, also the histopathological reports on the human and animal carotids.

Resumé of the Case Histories: Patient A. G., female, age 16 years. Patient was admitted to the Temple University Hospital, March 8, 1932, on the service of the senior author. On admission the patient presented the following: Bilateral purulent otitis media, right chronic mastoiditis purulent, and definite evidence of a septicemia. A blood culture was taken soon after admission, which was returned positive for streptococcus hemolyticus. Treatment of the

septicemia was immediately begun. Due to the poor condition of the patient operation was delayed. Five days after admission the patient presented definite signs of meningeal involvement, which was confirmed by a spinal puncture, and culture of the fluid yielded streptococcus hemolyticus. On this date the patient also presented evidence of embolic phenomena. Seven days after admission a radical mastoidectomy was done with exposure of the lateral sinus and the cerebral dura. At the same time the right common carotid artery was isolated and exposed for injection. The patient received three injections of 0.5 per cent neutral acriflavine and two injections of polyvalent antistreptococcus serum into the right common carotid artery. She also received numerous spinal punctures, intravenous medication, and whole blood transfusions. Her condition became progressively worse and the patient died ten days after admission. Permission for autopsy was not granted but we were able to remove section of the common carotid artery at the point of injection for histologic study.

We might comment on this case, stating that perhaps if operation had been done on admission to eliminate the focus of infection, which was undoubtedly in the mastoid, it might have aided. In any event it should be impressed that if the carotid arteries are exposed and utilized for injection purposes, both arteries should be used.

This was a proven case of streptococcus meningitis as evidenced by positive cultures from the blood, spinal fluid and mastoid wound yielding streptococcus hemolyticus.

Pathological Report (Patient A. G., slide No. 10058): The intima is practically normal, except that in one area a little thickening is noted. The internal elastic lamina may be seen as an unstained wavy line. An inflammatory process is seen in the adventitia, which shows hyalinization, hemorrhage and leukocytic infiltration, with equal proportions of mononuclears and polymorphonuclear leukocytes (not a suppurative process). The perivascular tissue also shows a certain amount of inflammatory reaction. In the focal area in the photograph the polymorphonuclear is the predominating cell

Patient M. C., female, age 6 years. Patient was admitted to the Temple University Hospital on the service of the senior author, March 22, 1932. The diagnosis on admission was acute right suppurative otitis media, acute right mastoiditis; bilateral sphenoiditis and ethmoiditis and meningitis, probably streptococcic. Immediately following her admission, a right simple mastoidectomy was done;

Fig. 1.

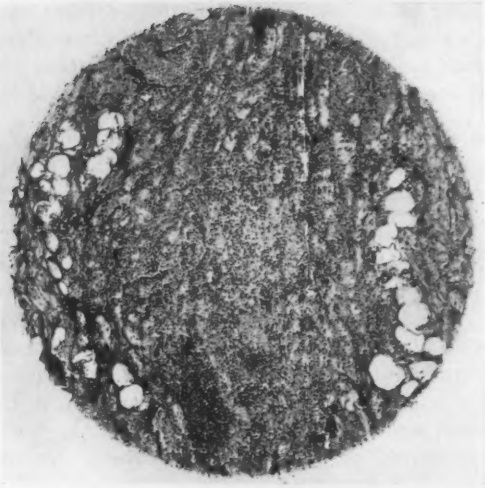

Fig. 2. Shows tissue infiltration and leukocytes.

bilateral ethmoidectomy and sphenoidectomy, and isolation and exposure of both common carotid arteries was performed.

Treatment consisted of spinal punctures done four times daily;

glucose and Pregl's iodine intravenously daily. Several injections of polyvalent antistreptococcic serum intravenously and also two injections of Dick's antistreptococcus scarlatinal serum intramuscularly. She also received at twelve-hour intervals five injections of neutral acriflavine, 0.5 per cent, two injections of Pregl's iodine and one injection of polyvalent antistreptococcus serum into the right common carotid artery and two injections of 0.5 per cent neutral acriflavine into the left common carotid artery. Despite this vigorous treatment, the patient became progressively worse and died six days after admission.

This was a proven case of streptococcus meningitis, as evidenced by positive cultures from the blood, spinal fluid and the mastoid wound yielding streptococcus hemolyticus.

Permission for autopsy was not granted but we were able to remove a section from each carotid artery at the point of injection for histologic study.

Pathological Report (Patient M. C., slide No. 10111): The intima seems in good condition. In one section in the tunica media is an area of necrosis. The adventitia shows hyalinization and necrosis, with extensive cellular infiltration—the polymorphonuclear predominating. In the perivascular tissue hyalinization with necrosis is also shown, with leukocytic infiltration almost approaching abscess formation. In one area in this specimen a definite tract can be seen, possibly the line through the needle has been introduced.

Patient M. S., female, age 26 years. Patient was admitted to Temple University Hospital on Feb. 26, 1932, on the service of the senior author. On admission to the hospital, patient was bright and cheerful, able to walk about, and no signs of meningeal irritation. Patient presented on examination a chronic suppurative otitis media on the right side, which was of fourteen years' duration, and a chronic mastoiditis. Four days after admission a right radical mastoidectomy was performed. Following the operation, the temperature, which up to this time had been normal, rose and remained elevated. The patient was stuporous. Examination revealed signs of meningeal irritation, confirmed by spinal puncture which yielded streptococcus hemolyticus on culture. Three days after the first operation, the patient was taken to the operating room and the mastoid wound reopened. This was followed by a release of considerable pus; a decompression of the middle fossa was done with incision of the dura and exposure of a temporal lobe abscess. At this time

Fig. 3. Hemisection of common carotid shows hemorrhagic infiltration.

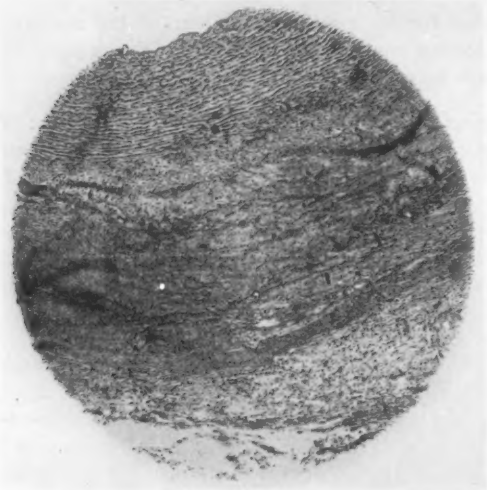

Fig. 4. Close-up showing area of hemorrhagic extravasation.

exposure of the carotid arteries was done. Treatment consisted of intravenous medications and several blood transfusions; the patient also received intracarotid treatment, consisting of 0.5 per cent neutral acriflavine and Pregl's iodine into the carotid arteries. Patient became progressively worse and died, March 6, 1932, ten days after admission. Autopsy revealed a temporal lobe abscess, which had ruptured into the right ventricle of the brain. Section of the right carotid artery was removed for histologic study.

This was a proved case of streptococcus meningitis otitis in origin as evidenced by positive culture yielding streptococcus hemolyticus from the blood, spinal fluid and the mastoid wound.

Pathological Report (Patient M. S., slide No. 9987): Examination of this specimen microscopically reveals no pathologic changes either in the tunica media or adventitia; the intima is also normal.

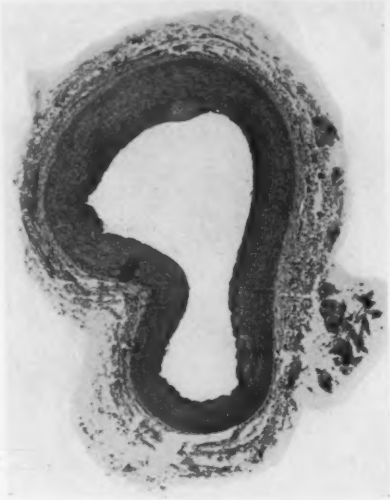

Fig. 5.

Case Report of Dog: The following two sections obtained from a series of dogs illustrates definite damage to the vessel. In these animals we purposely used heavier dull needles and injected the solutions rapidly. In exposing the vessel and inserting the tapes, excessive trauma was inflicted on the vessel. This was done in order to study the amount of trauma the carotid could withstand before extra and intravascular damage is done.

Pathological report of vessels obtained as a result of animal experimentation follows:

Microscopic Examination (section from right common carotid of dog): Vessels on the right side in the experimental dog shows a most intense periarteritis with a very great number of polymorpho-

nuclear elements invading the outer coat of the vessel wall, and with a tendency towards necrosis of tissue. There was an invasion of blood elements and a secondary fibrosis. No change could be made out in the intima.

Fig. 6. Dog. Right common carotid.

Microscopic Examination (section from left common carotid of dog): On the left side the involvement of the vessel wall is much less marked as regards the inflammatory elements in the outer coat. Here, however, there is an intense edema and invasion by some inflammatory elements. The striking thing is the denudation of the intima and the tendency to a well organized clot at one side of the vessel wall. One can very easily see that in a vessel of this sort there would be eventually a complete closure of the lumen by a thrombus.

Having determined the actual pathology of the blood vessels resulting from repeated intracarotid medications, we concluded the following:

a. The vessel may be injured externally.

b. The vessel wall itself may be weakened.

c. The intima may be traumatized because of careless penetration of the needle, which was either dull or too large.

Because of the queries in regard to the changes taking place in the carotid after repeated intracarotid injections, we should like to digress somewhat, and review the technique of carotid exposure and the method of injection. Although the method of exposure very nearly resembles the standard description of the jugular ligation, still there are a few salient points and precautions that we should like to emphasize as they have some bearing on the subject.

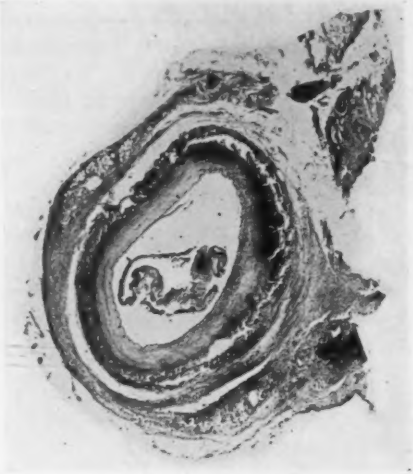

Fig. 7. Dog. Left common carotid shows thrombosis.

DESCRIPTION OF OPERATION.

The neck is prepared and sandbags are placed under the shoulders. The neck is extended and face is turned to the opposite side. The position of the common carotid artery is indicated by a line drawn from the upper part of the sternal end of the clavicle to a point mediary between the tip of the mastoid process and the angle of the mandible. From the clavicle to the upper border of the thyroid cartilage this line overlies the common carotid; beyond this it is over the external carotid. An incision is made beginning at the thyroid cartilage, along this line and extending downward about 8 cm. The

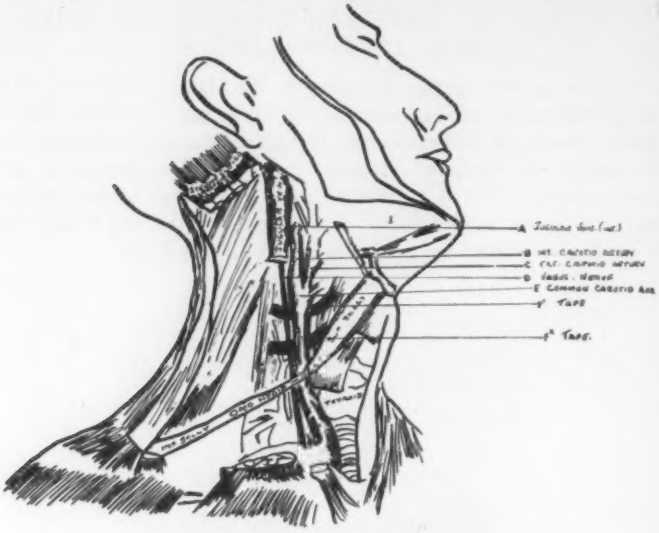

Fig. 8.

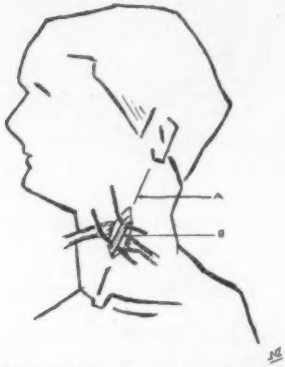

Fig. 9. Shows wound edges retracted, common carotid artery exposed and held in view for injection by two traction guides of umbilical tape.

skin, superficial fascia and platysma are incised and retracted, and ribbon muscles are retracted. After the incision is made, the face is turned upwards. The anterior border of the sternocleidomastoid muscle is then retracted laterally. This is an important step for if the muscle is not retracted, often it is very difficult to locate the

artery which lies under the muscle. By palpation the artery is located and the sheath is isolated and incised, exposing the carotid artery lying medially, the internal jugular vein laterally and the vagus nerve posteriorly and between. An aneurysm hook is then passed beneath the common carotid artery, using great care not to injure the fascial investment of the carotid, avoiding blunt dissection. Two pieces of umbilical tape are first soaked in sterile mineral oil and then threaded on the aneurysm needle and passed beneath the vessel above and below the point of injection. These are used as traction sutures and remain in situ (see Fig. 8). The portion of the artery to be injected is then drawn into view by making traction on these sutures, using a 23-gauge, 1½-inch hypodermic needle and a 10-15 cc. Luer syringe, preferably with a safety lock. The needle is carefully inserted into the vessel and blood is drawn back into the syringe. The injection is given slowly and after the needle is withdrawn it is followed by surprisingly little bleeding. The wound is flooded with sterile mineral oil, then a small piece of vaseline gauze dressing is placed into the gaping wound and the two pieces of umbilical tape are tied loosely over the gauze and then another gauze dressing and bandage are placed over all. When it is desired to repeat the injection, under sterile precautions, the dressing is removed and the tapes are loosened, and the piece of gauze is removed. Then with sterile gloved finger, the new adhesions are carefully broken up and the vessel is brought into view (see Fig. 9) by traction on the tapes and the injection is repeated as described.

Following the above technique, we have been able to administer serum, glucose, Pregl's iodine and neutral acriflavine without injury to the vessel, as proven by Case 3, where the precautions were religiously observed.

COMMENTS.

It is common anatomical knowledge that besides the carotid sheath the carotid itself is surrounded by a thin fascial investment. As mentioned in our pathological reports, a periarteritis with inflammatory exudate is often produced by manipulation, needle puncture and traction.

This fascial investment plays an important role in the protection of the vessel. It is made up of connective tissue and it is through this structure that the vasa vasorum are carried to the carotid and then enter into the outer coat of the vessel, known as the adventitia. Accompanying these vessels are the lymphatics.

We observed that whenever we protected the fascial layer during carotid exposure, on postmortem there were no gross pathological changes in the vessel walls. On the other hand, rough handling and stripping of the sheath produced a periarteritis, cloudy swelling and areas of necrosis. We concluded, therefore, that by unnecessary manipulation, we disturb the vasa vasorum, thus interfering with the nourishment and the circulation of the vessel wall; the histological changes noted are the result of a circulatory disturbance as much as a mechanical factor.

Assuming that we have preserved the carotid fascia during the carotid exposure, we must still be careful not to injure unnecessarily the sheath when the carotid is lifted from its bed. This is performed with an aneurysm needle and two pieces of umbilical tape, which are inserted underneath the artery. The umbilical tapes are used for the purpose of traction so as to bring the artery to the surface whenever it is necessary to inject. It might be well to mention that by soaking the umbilical tapes in sterile mineral oil, we avoid unnecessary friction and traction. Thus, we further save the fascia, prevent injury to the vasa vasorum and eliminate any possible periarterial irritation.

After each treatment, we made it a steadfast rule to fill the wound with sterile mineral oil so as to prevent rapid formation of adhesions and fibroconnective tissue; as during the interim an exudate which is fibrinous in character is thrown around and about the vessels, and the surrounding tissues adhere to the external surface of the artery. When repeating the treatment, by carefully separating these new adhesions with the gloved fingers, one must use great care not to tear away the adhesions from the surface of the vessel; thus, we will further avoid injury to the vessel wall.

From practical experience, we learned that the needle and its mode of introduction plays an important role in intracarotid therapy. For that purpose, we employ a sharp 23-gauge, 1½-inch needle in order to avoid tearing the wall or disturbing the intima. When introducing the needle, the beveled edge should point upwards. One should eliminate all unnecessary stabs and never repeat the injections at the same point, otherwise atheromatous areas and a possible aneurysm may develop.

Very often one fails to inject properly, due to the fact that the vessel changes position with inspiration and expiration. In several instances the needle point penetrated the vessel wall and the outer surface of the vessel became infiltrated and some of the fluid ex-

travasated into the tissues. This also occurred when the needle punctured the inner wall. It injured the intima, and produced an infiltration on the inner surface of the vessel without extravascular extravasation. Whenever this occurred, invariably a thrombosis resulted.

For some unknown reason the circulating blood will not coagulate when passing through a vessel whose endothelium is intact. However, when the endothelium is injured, the blood will clot and produce a thrombus. A similar result may be produced when the medicaments are injected improperly. With this in view, the injections of the drugs into the carotid should be given slowly in order not to interfere with the blood current in the artery.

For a time we thought that the drugs were responsible for the thrombosis, but we determined that it was not the contents injected that were causative but it was the method of injection, and the rapidity with which the fluid was instilled.

Normally, there should be no difficulty in forcing the medicaments out of the syringe. However, there are times when the needle enters into the vessel wall and there is no return of blood into the syringe when the piston is drawn back; when this occurs the injection should not be continued. If we reintroduce the needle at another point, and we are reasonably sure that we are in the lumen, and if the blood still cannot be drawn back into the syringe, then we may assume that a thrombosis is present.

We also observed that when extreme traction was produced, a kink was formed in the vessel. Therefore, when the needle is inserted it is best to avoid the vascular kink. Otherwise, the needle will penetrate the inner wall of the opposite side of the vessel at the maximum point of the kink. When the kink is produced, we narrow the lumen, retard the circulation and traumatize the intima of the vessel and expose ourselves to the danger of a thrombosis.

Before closing, we wish to mention several facts which were called to our attention following a discussion with Dr. O. V. Batson.

We noted, in one of our cases, that following the injection of 0.5 per cent neutral acriflavine (see illustration, Case M. C.) there was staining of one-half of the head and neck within a very short interval after the injection was given, and there also was a staining of the conjunctiva. It is the first flush of the dye that is important, for it shows that the dye is being brought into immediate contact with the brain and meninges, even though only for an instant before

it is carried into the general circulation. To quote from Dr. Kolmer's article ("Chemotherapy and Serum Treatment of Pneumococcus and Streptococcus Meningitis," *Archiv. Otolaryngol.*, 9-1929-1, p. 509, April): "There is intense staining of the meninges, cerebral and basilar, following the injection of the dye into either or both common carotid arteries, with the staining of the cerebral spinal fluid.

"Injection into the internal carotid arteries is preferred but operative difficulties are considerable. So that injection into the common carotid arteries have been used almost entirely, even though a part of the inoculum is lost or wasted, as it were, through the external carotid artery."

In view of this fact, we wish to stress the suggestion made by Dr. Batson, that a clamp of some kind be placed on the external carotid artery so that even though the injection is made into the common carotid artery, all the dye will pass through the circulation of the internal carotid artery. To modify our description, it might be well to make our initial incision at a higher level, closer to the bifurcation and while passing the tapes beneath the common carotid, we should also pass a tape of a different color beneath the external carotid, so that it, too, can be drawn into the wound, and by some means compressed while the injection is being given.

The internal carotid supplies the anterior portion of the brain and the posterior portion is supplied by the vertebrals which are branches of the subclavian and which unite to form the basilar. It would be advisable in otitic meningitis to utilize the vertebral for injection purposes. (It might be well to mention that to locate and inject the vertebrals is not an easy task; we have had the opportunity to watch such an injection being given in a case of pneumococcus meningitis.)

Another point worth while mentioning is that in cases of meningitis following sinus infection, where carotid therapy is used, it is essential not to interfere with the circulation of the external carotid artery.

It is also a known fact that whenever we bring into view a blood vessel during the course of blunt dissection, as in isolating and exposing the carotid vessels, we have really removed from it the periarterial tissue and the adventitia; thus, we actually are performing a periarterial sympathectomy, and this is accompanied by a primary dilatation of the blood vessels.

CONCLUSIONS.

We believe that intracarotid therapy offers a ray of hope in the treatment of an almost fatal complication.

We believe we have developed a technique which will minimize extra and intravascular changes, especially thrombosis of the vessel, atheromatous changes and aneurysm.

We are of the opinion that the intima suffered less injury than any other portion of the artery; the media revealed areas of necrosis, hyalinization and infiltration with various cells.

The adventitia seemed to bear the brunt of pathologic changes, showing necrosis, infiltration and almost approaching abscess formation.

Both carotid vessels should be utilized in order to bring the medicaments in contact with both sides of the brain.

The arteries can stand a reasonable amount of trauma provided considerable care and caution is exercised.

There is no doubt that a certain amount of damage is done to the carotids following intracarotid therapy; however, this should not deter anyone from employing this method in the treatment of meningitis.

In closing, we desire to express our sense of indebtedness to Dr. Stanley Seigle and Dr. Sidney Shapin for their conscientious aid, and to Dr. N. W. Winkelman and Dr. Frank W. Konzelmann for their collaboration in the interpretation of the pathological tissues.

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BACTEREMIA OF OTITIC ORIGIN, ROUTES OF INFECTION TO THE BLOOD STREAM; CASE REPORT WITH RECOVERY.

Dr. Samuel D. Greenfield, Brooklyn.

Otitic infections may give rise to a bacteremia through the following pathways: *1.* By direct extension of the suppurative process from the middle ear, through the mastoid cells by continuity, to some portion of the lateral sinus and thence to the general circulation.[1] This is by far the most common route. *2.* By direct extension of the infection through the floor of the middle ear to the jugular bulb and thus to the blood stream. This may be by way of persistent vascular channels in the bony roof of the bulb,[2] or through dehiscences in the floor of the middle ear cavity. The infection takes this avenue most often in infants and young children, giving rise to the so-called primary bulb thrombosis. *3.* By primary involvement of the mastoid emissary vein. The latter in its course to join the sigmoid portion of the lateral sinus may traverse infected para- or postsinal cells. The micro-organism may gain access to the general circulation either directly through the emissary vein or by extension of the thrombophlebitis from the vein to the lateral sinus and then enter the blood stream. This is more apt to occur when the mastoid emissary vein is of large size.[3] *4.* By following upon a thrombosis of the inferior petrosal sinus. This may result from extension of disease from the labyrinth by way of the veins of the internal ear, or directly from necrosis of cells in the apex of the petrous pyramid.[4] *5.* By primary involvement of the superior petrosal sinus. This results from extension of disease from the middle ear and antrum through the veins which pierce the tegmen tympani and antri. These veins pass through the petrosquamous suture and join the veins of the middle ear mucosa and empty into the superior petrosal sinus.[5] *6.* Through other diverse channels leading from the mastoid cavity which heretofore have gone undetermined. These avenues along which micro-organisms can travel

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may consist of anatomical variations or anomalies in the form of dehiscences in the bone or persistent vascular channels, to which our attention has been called by Wittmaack. On the other hand, the organisms may follow the course of the normal preformed body openings of the diploic vessels, the emissary venules and, lastly, the lymphatic system. That there are pathways other than those above described and already well known which may carry infection from the mastoid cavity to the blood stream without intermediate involvement of any other large venous tributary is fairly well demonstrated in the case I am herein reporting. To prove my contention conclusively one would require postmortem histopathologic evidence. But this patient recovered and such authentic and irrefutable data is therefore not available. Nevertheless close analysis of the subsequent clinical course after operation in this particular case tends to offer within reasonable certainty the contention that the infection from the broken down mastoid cavity gained access directly to the blood stream through some route other than the lateral sinus, the jugular bulb, the mastoid emissary vein, the superior petrosal or inferior petrosal sinuses. At operation this patient presented a badly broken down mastoid from which source organisms were making their way into the general circulation. The lateral sinuses were both normal. The mastoid emissary veins were not involved. It is reasonable to suppose also that the bulb and the petrosals were free from infection because immediately upon liberating the pus and draining the mastoid process the blood culture became negative and the temperature remained down. This would not and could not be the case if any of the larger venous tributaries had been the intermediate seat of infection from which the blood stream was receiving organisms.

I should therefore like to record this case as one in which a bacteremia resulted from the mastoid abscess through some route other than those that have heretofore been described.

Case Report: S. B., age 7 years, was admitted to the Beth Moses Hospital on April 9, 1932, complaining of temperature, malaise, a discharging right ear and headache.

Previous Personal History: The patient had measles and whooping cough when 3 years of age. One year after he had a tonsilloadenoidectomy performed. Following a grippe infection several months later the patient developed an acute suppurative otitis media (left). The drum was incised and after two weeks of otorrhea and temperature a diagnosis of an acute surgical mastoid was made and

the child was operated at the Beth Moses Hospital. At this time the mastoid was thoroughly dissected and according to the operative record there was no exposure of sinus or dura. The patient remained in the hospital one week and after being afebrile for three days he was discharged. The postoperative course remained uneventful and the child was well after five weeks. The middle ear was dry and the posterior wound was firmly healed. The patient remained comparatively well until three weeks before admission to the hospital.

History of Present Illness: On March 20, 1932, the patient suffered with an upper respiratory infection. He developed a severe sinusitis and a cough. On the third day of his illness the patient experienced pain in the right ear. He had a temperature of 104° and complained of severe headache. The next morning the right drum ruptured spontaneously and the middle ear began to discharge freely. The pain in the right ear abated completely and the discharge which at the beginning was profuse, lessened in amount considerably by the end of the week. For several days after the drum ruptured the temperature showed some tendency to come down but four days prior to admission to the hospital the patient complained of chilly sensations and the temperature rose to 104° and 105° daily. He complained of headache but had no sign nor symptom referable to the left ear. He seemed apathetic and slept most of the day. He was admitted to the Beth Moses Hospital for observation and diagnosis.

Admission Notes Read as Follows: Temperature, 104.4°; pulse, 128; respiration, 28. Patient is a young male child, whose general condition is fair. There is marked pallor. The heart is rapid, with good muscular quality and absence of murmurs. The lungs are negative and the abdomen discloses no masses, no tenderness and no palpable spleen. There is absence of skin eruptions and no petechiae are visible. The extremities are negative. All reflexes are normal and there is absence of neck rigidity, Kernig and Babinski reflexes. Otolological consultation advised.

Rhino-otological examination by Dr. Greenfield, April 9, 1932: The nasal mucosa is congested with the presence of some mucopus on the floor of each nasal chamber. The throat is congested; the tonsillar fossae are clean. Right ear: There is absence of mastoid tenderness. The aural discharge is scanty and thin, without odor. The drum membrane is flat and there is no evidence of retention in the middle ear. The canal wall is normal. Hearing shows whispered

voice at five foot distance. Left ear: There is absence of tenderness over the old mastoid scar, the latter is pale, showing no redness and no edema. The discharge in the canal is scanty and thin, without odor. The drum membrane is flat, with all landmarks visible. There is a small central perforation. The hearing shows whispered voice at eight feet.

Opinion: With a temperature of a septic nature and a history of a recent acute otitis one must bear in mind the possibility of a blood stream infection.

Clinically neither mastoid presents sufficient evidence to warrant surgical interference. Advise blood culture, blood count, urine examination and X-ray of the thorax.

April 10, 1932: General condition of the patient is unchanged. The temperature dropped to 103.2°. The following are the laboratory data received: Blood count: hemoglobin, 56 per cent; R.B.C., 2,930,000; W.B.C., 11,000; polys., 65 per cent; lymphs., 35 per cent. Urine examination negative except for a slight trace of albumin and an occasional pus cell. X-ray of the thorax discloses nothing of pathological significance. X-ray of the mastoid showed the following: right mastoid, slight haziness of all mastoid cells; sinus visualized with anterior limit 1.25 cm. from posterior canal wall. There is no evidence of bone softening. Left mastoid characterized by absence of all cell structure. The anterior sinus wall very sharply outlined at about 1.25 cm. behind canal wall. Blood culture again taken.

April 11, 1932: The general condition remains unchanged. The temperature dropped in the morning to 100° and at noon following a series of definite chilly sensations, it quickly rose to 105°. The blood culture, taken on the ninth, was reported positive for streptococcus hemolyticus. The one taken on the tenth was also reported positive for streptococcus hemolyticus. With two positive blood cultures, chilly sensations, a temperature that was typically septic, a marked anemia, there was no question we were dealing with a bacteremia. The problem that presented itself was the question as to which ear was the offending one. There was nothing in the otoscopic pictures or the otitic signs or symptoms to indicate from which ear or mastoid the organisms were gaining access to the general circulation. The procedure decided upon was to first operate on the old mastoid.

Operation, April 12, 1932: The old scar was incised and the soft

parts retracted. A considerable portion of the cortex had regenerated. After removing this I was very much surprised to find that the mastoid was completely excavated and filled with thick creamy pus from which the laboratory subsequently isolated a pure culture of streptococcus hemolyticus. The cavity was limited by what appeared to be normal sinus and dural plates. The sinus was uncovered from the knee to the tip and found to be normal. The right mastoid was then opened, the cells showed no softening. They contained very little exudate. The sinus and dural plates were unbroken and appeared to be free from disease. The lateral sinus was uncovered widely and found to be normal in color and texture. With these operative findings it was deemed advisable to do nothing further. The right wound was sutured in the usual manner but the left one was permitted to remain widely open and packed lightly with iodoform gauze. The patient left the operating table in good condition.

April 13, 1932: The temperature dropped in the morning to 99° but rose in the evening to 101.6°. The patient was comfortable and complained of slight pain in the left ear. A transfusion of 250 cc. was given by the direct method.

April 14, 1932: The general condition of the patient was very much improved. The temperature dropped in the morning to 99° and rose in the evening only to 101°. A blood culture was taken and subsequently reported negative. The patient's outer dressing was changed.

April 15, 1932: The patient's temperature reached normal in the morning and rose in the evening only to 100.2°. His general condition was very much improved. He seemed brighter and asked for food. There was no question that his infection was well checked and that the blood stream was no longer receiving a fresh supply of micro-organisms.

April 16, 1932: The patient was now well on the way to recovery. His dressing was changed and his right wound was healing by primary union. The left mastoid was clean. The temperature was normal all day. A blood count showed the following: Hemoglobin, 70 per cent; R.B.C., 3,700,000; W.B.C., 16,000; polys., 67 per cent; lymphs., 33 per cent. From this day on the patient was afebrile. He remained in the hospital for one week longer and was discharged May 23, 1932, well on the road to recovery. He continued dressings until June 10, 1932, at which date he was discharged cured with firmly healed posterior wounds and dry middle ears

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A CASE OF MASTOIDITIS ASSOCIATED WITH HEMATURIA.*

Dr. Abram H. Persky, Philadelphia.

Before presenting this case I would like to offer an explanation for the title of my paper. In a sense it may be a misnomer for the case was originally purely that of an acute suppurative mastoiditis. In fact, this was a secondary mastoiditis, for the present infection had been superimposed upon a previous mastoidectomy. The discovery of the symptoms of hematuria and a coincident nephritis was accidentally made by the nurse in charge, as she noticed that the diapers were bloodstained. The association of an infection in the tonsils is also an important factor in this case and should perhaps also have been included in the title. However, for the sake of brevity I have limited my title to "Acute Mastoiditis Associated with Hematuria."

There has recently been an abundance of papers dealing with either hematuria or acute hemorrhagic nephritis. It is not my intention of merely adding another case report to this collection. On the other hand, there are enough significant features in this case to warrant a resume and discussion of these various factors. We know that the symptom of hematuria in children occurs not at all infrequently in the course of any severe infection. Most any conceivable disease has been discussed as a causative agent. We know also that follicular tonsillitis is an extremely frequent culprit. We have a series of cases in another hospital of hematuria, or acute hemorrhagic nephritis, if you wish, traced directly to acute sinusitis in children; recovery of the condition only occurring after radical surgical treatment of the sinuses. However, there are comparatively few cases where an infection in the middle ear, or an acute mastoiditis, particularly of the so-called hemorrhagic type, acts as a causative agent in the production of hematuria.

That the acute mastoid infection may act as a focus of infection to the rest of the body has been stressed enough recently in detail not to burden you with this repetition. You are all familiar with

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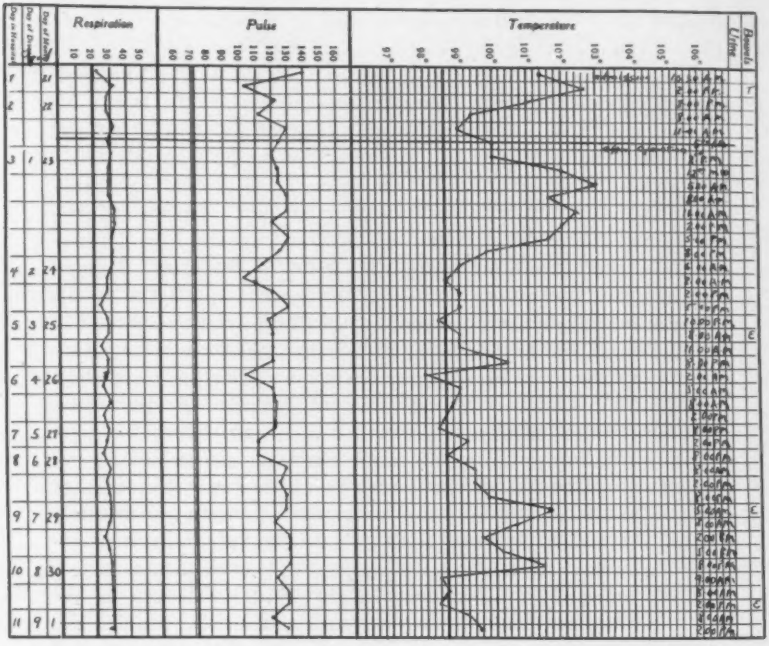
the work of Lyman and Alden, who showed the direct relationship between a streptococcic hemolytic infection of the mastoid and the gastrointestinal disturbances of infants, characterized particularly by diarrhea, vomiting and loss of weight. L. W. Dean, in 1927, also did a splendid piece of work along the same lines. Renaud, in 1921, concluded that infants suffering with the gastrointestinal syndrome are doing so because they are infected and they should be treated first for the infection. His second conclusion was that the ears of the infants act extremely frequently as a causative agent. His conclusions can just as well apply to the cases of acute hemorrhagic nephritis. I can continue and give observation after observation from the literature as to the incidence of infection in the ears on systemic disturbances in children. I might cite the article of George M. Coates, who showed direct relationship between the infected ear and gastrointestinal disturbances. The principles underlying this infection apply just as well to those underlying acute hemorrhagic nephritis. Dick's conclusions are extremely interesting. He showed definitely that where the migration of bacteria from the foci of infection takes place, the bacteria are often found in the urine. I might also add the observations of S. J. Crowe, who reports the results of tonsillectomy in eighteen cases of nephritis; four of these had mitral disease and two had also had chorea. Following the tonsillectomy, in the majority of cases the urine cleared up completely. Loncope, in a study of forty cases of acute and subacute glomerular nephritis, found that tonsillitis occurred in 53 per cent of the cases and that the hemolytic streptococcic organism occurred in 81 per cent of the infections. Ersner's report on three cases in a series of eighty-one mastoid infections that were associated with acute hemorrhagic nephritis, or hematuria, also points out the infective principles involved. There was a hemolytic streptococcic organism isolated in the mastoid. He concludes that the mastoid can be the active cause of infection and the hematuria, and contrawise that the persistent hematuria associated with mastoiditis should be regarded as another positive indication for mastoidectomy.

N. L., age 2 years, was admitted to the Northern Liberties Hospital on April 21, 1932, with a history that over two weeks ago he suddenly became feverish, irritable, began to sleep poorly and would not eat. Three days after the onset of this illness, his left ear was incised and it has been discharging ever since.

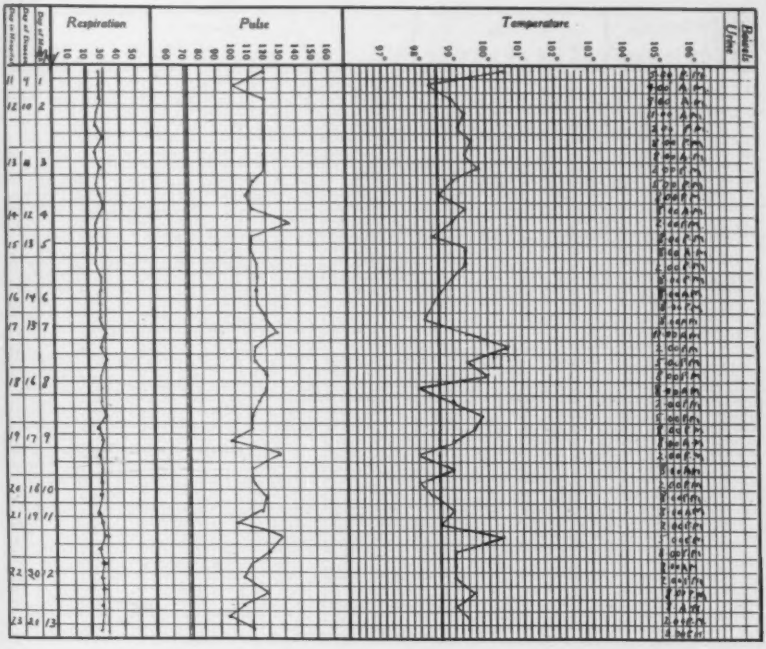
P. M. H.: When this child was 8 months old, he was admitted to the Graduate Hospital, where a bilateral mastoidectomy was per-

formed. At that time there was cavity formation and free pus in both mastoids. The pathology of the two sides was identical. The organism isolated was a streptococcus hemolyticus. At that time he made an uneventful recovery and was discharged from the hospital eight days later. During his stay in the hospital three urine specimens were examined and on each occasion a normal urine report was given.



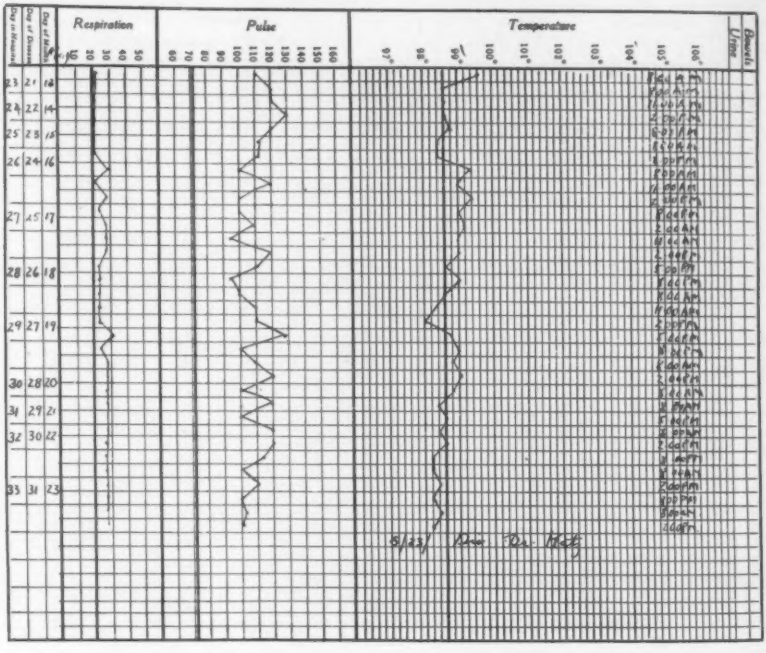
On readmission to the Northern Liberties Hospital, there were unquestionable symptoms of an acute mastoiditis and the day following his admission a left mastoidectomy was performed under a general anesthetic. Free pus, necrosed bone and granulation tissue were found in the mastoid at the time of operation. The organism isolated was again the streptococcus hemolyticus. On the day of operation the urine analysis revealed a +2 albumin, 20 W.B.C. and 15 R.B.C. His blood count was 3,750,000 R.B.C., Hb. 70 per cent, polys. 60 per cent, small lymphs. 38 per cent. He had a normal

reaction and apparently was doing well for two days, and then became irritable, fretful and his temperature began to rise to 103°. Following this, he continued to have an irregular temperature, going up for a few days and then down for a few days, as the chart will indicate. During this period the nurse began to notice that the diapers were somewhat pink stained, and on the sixth day after the operation, another urine analysis revealed a +2 albumin, 8 W.B.C., and un-



countable R.B.C. This seemed to account for his general uneven convalescence and sickly appearance. Apparently the nephritis at the time of operation was lost sight of and only brought back to our mind by the general stormy convalescence. Following this a urine analysis was examined every other day. Throughout this period and up to the day of discharge there were variations in the picture, but there was always a certain amount of albumin present and always a few W.B.C., and while at the outset the R.B.C. could not be counted, yet at the time of discharge there were still 5 to 10 present

in his specimen. In addition there was always a secondary degree of anemia, for on May 5, 1932, the blood count revealed 3,450,000 R.B.C., 13,400 W.B.C., 54 per cent Hb., 59 polys., 41 small lymphs. At the time of the discharge, Dr. Melman and myself felt that this was a case of acute hemorrhagic nephritis due to a streptococcic infection having two foci, one in the mastoid and the other in the tonsils. In view of this we recommended a tonsillectomy to elim-



inate the second focus, for the mastoid wound was clean and practically healed. The child was discharged on the twenty-third, but returned two days later, on the twenty-fifth, when a tonsillectomy was performed, and he stayed in the hospital until June 5, 1932. Throughout this time there was still a slight elevation of temperature, but other than that, and the findings in the urine, he appeared to be perfectly normal, playful, eating well, sleeping well and apparently gaining weight. The urine analysis on three occasions, while in the hospital, continued to show 1 to +2 albumin and occasional

W.B.C., and anywhere from 5 to 10 R.B.C. This child has been under observation in the dispensary where Dr. Melman has been seeing him at irregular intervals and apparently there is a marked improvement in the entire condition. The last few reports of the urine analysis still showed a +1 albumin and a few W.B.C. (4 to 5), but no R.B.C.

In reviewing this case I wish to state that it has not been presented with any idea of adding to the already voluminous amount of literature on this subject, but more for the purpose of presenting several interesting features: first, a case of acute suppurative mastoiditis, which was in itself a secondary infection. This raises the question of re-infection of the mastoid that had already been operated upon. Unquestionably the relation of the infected tonsil has played an important role in this re-infection of the mastoid; second, the focus of infection, both in the mastoid and in the tonsils, giving a series of extraneous symptoms, in our case localized in the kidneys. Three, the amelioration of the symptoms by the removal of one focus of infection and the practical cure by the removal of the second.

Conclusions: 1. A case of an acute hemorrhagic nephritis caused by streptococcus hemolyticus infection, which has foci at two points, one in the mastoid, and second, in the tonsils.

2. A secondary mastoiditis produced by the same organism that caused primary infection, presumably due to the diseased tonsils. As to whether the removal of these tonsils will prevent any future attacks of mastoiditis is problematical, although statistics show that re-infection is unquestionably more common where the tonsils have not been removed.

3. A tonsillectomy is of prime importance in cases of hematuria, or acute hemorrhagic nephritis, and should be considered even in spite of the presence of albumin or R.B.C. in the urine.

4. The significance of a primary focus—the tonsils—activating a secondary focus—the mastoid—which in turn aggravated an underlying *systemic condition*—in our case an acute hemorrhagic nephritis.

1922 Spruce Street.

RHINOLITHIASIS; REPORT OF THREE CASES.*

Dr. Jos. M. Polisar, Brooklyn.

Due to the more frequent and routine examination of patients, large rhinoliths are rarely encountered today. In neglected cases, however, they may reach the weight of 85 gm. or more. The largest concretion recorded, which was as large as a hen's egg and weighed 110 gm., destroyed the septum and turbinates and required a Rouges operation (sublabial-rhinotomy) for its removal.

According to Sir St. Clair Thomson[1] more than 300 cases of rhinolithiasis have been reported. Since then, F. E. Locy,[2] of the U. S. Navy, reported a case where the rhinolith measured 0.75 cm. and had to be broken up with a heavy forceps in order to remove it. Another case, reported by A. P. Jana,[3] where the calculus was the size of an adult's thumb, also had to be crushed in order to facilitate its removal. An interesting case reported by M. C. Myerson,[4] where a rhinolith required an external nasal operation for its removal. Still another case by Jay Ireland,[5] where a patient complained of complete nasal obstruction on the left side of the nose for over sixteen years, caused by a rhinolith which, after extraction, showed the structure to be definitely laminated.

Nasal calculi are usually unilateral, generally spherical and irregular. Some show prolongations; they are friable with a rough mulberry-like surface and of a gray or brownish-black color. Qualitatively, they consist of calcium phosphate and magnesium phosphate, sodium chloride and calcium carbonates and organic bodies of mucus and protein.

These salts originate from the nasal mucus to some extent, but still more so from tears. On section it is found that the salts have been deposited around a nucleus of some foreign body, such as fragments of inspissated mucus, clots of blood, sequestrum from the septum or turbinates, small beads, nutshell, etc.

If a foreign body acts as a nucleus it may have been introduced through the anterior nares during vomiting or choking, or it may have

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penetrated the tissues as in the case of lead bullets or particles of stone or metal during an explosion.

A true rhinolith is one formed around a nucleus of blood or mucus. It is rare before the fourth year. A false rhinolith is one in which a foreign body becomes coated with salts. It may occur at any age. Some have been known to have remained in position for a period of thirty years or more.

The symptoms of rhinolithiasis may vary from a slight one-sided discharge or nasal obstruction to grave structural changes. Fetid discharge, hemorrhages or obstruction to breathing are symptoms common in most cases. Deviations of the septum to the opposite side and destruction with prominence of the molar bone have been described by Baber. Knight mentions perforations of the palate and facial paralysis as having occurred. Disturbances of smell and middle ear disease are sometimes occasioned by these obstructions. The diagnosis may be easy or difficult, depending on the size or position and secondary changes which it may have occasioned. It should be differentiated from caries, osteoma and malignant growths. Probing the posterior nares through the pharynx as well as the anterior nares will help materially.

Prognosis: Once completely removed there is no tendency for nasal calculi to reform.

Treatment: Depends upon the size and extent to which the stone is embedded in the surrounding tissue and the structural changes it might have caused. Some may require a general anesthetic for its removal.

CASE REPORTS.

Case 1: Mr. A., age 23 years, a lawyer, consulted me in August, 1927, on account of right-sided nose bleeds, right unilateral headaches, right nasal obstruction and subject to colds. All of these alternately or conjointly for the past four years.

Examination revealed the left nasal passage to contain a moderate amount of mucus discharge and a slight deviation of the septum high up. The right nasal chamber presented evidence of recent bleeding, as shown by dried blood crusts. To facilitate the removal of the crusts a solution of 5 per cent tutocaine with adrelolin chloride was sprayed into the right nasal chamber, followed by a cotton applicator saturated with 15 per cent ichthyol in glycerine. The softened masses were removed by suction. Here the suction cannula encoun-

tered a firm, unyielding mass, which gave the sensation of bare bone or porcelain. This mass seemed to fill the entire right nasal chamber from about the middle anteroposteriorly, thus firmly encroaching upon the septal and lateral surfaces, as well as the floor and supe-

Fig. 1. Enlargement of the Indian nut nucleus showing the superimposed irregularities.

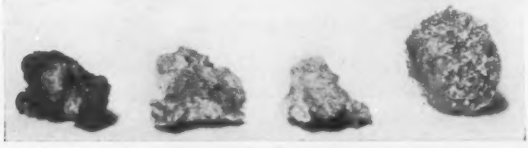

Fig. 2. Actual size.

riorly. The right middle turbinate was entirely obstructed from view. The inferior turbinate was compressed from above downward. Probing also met with resistance throughout and any firm pressure with the probe or on slight manipulation with nasal forceps caused excruciating pain and slight bleeding on the septal side. The mass presented the appearance of irridescent iron ore, dirty greenish

with tan irregular lines and a shiny metallic lustre at places. A similar appearance was revealed by posterior rhinoscopy; the mass protruding into the posterior nasal chamber and seemingly resting on the rostrum of the sphenoid.

X-ray (258) showed cloudiness of both antrums, the right more than the left. An opaque area at the midpoint of the nasal chamber is noted.

A diagnosis of rhinolithiasis was made. This was confirmed by crushing and removing a small portion through the anterior nares for examination. Removal under general anesthesia was advised. The operation presented some difficulty inasmuch as I did not want to crush the calculus and because of the resistance and profuse bleeding encountered when attempting to remove the stone through the anterior nares. I decided to push the mass through the anatomically larger posterior space and into the pharyngeal vault. To avoid aspiration or swallowing of the mass or particles thereof, I employed a long teaspoon as a tongue depressor, with its bowl end against the posterior wall of the pharynx to catch the mass. There was no necessity for it, however, as the mass was firmly held in the pharyngeal vault. It was easily grasped and removed with nasal forceps, followed by very little bleeding. Within ten days there was complete healing. There was practically no reaction, his headache ceased the day of the operation and he had no recurrence since. The mass weighed 53 gr. It was the size of a hazel nut, measuring 2.54 cm. x 1.95 cm. x 1.95 cm. On closer examination the tan lines on the stone proved to be particles of an Indian nut shell, which had evidently served as the nucleus of the rhinolith.

Case 2: S. F., student, white, age 14 years. This boy came to my office on Oct. 26, 1929, complaining of frequent frontal headaches, profuse sanguinopurulent discharge from the left nostril and postnasal discharge, especially at night. He was subject to frequent colds and nose bleeds on the right side. At the age of 5 years he was injected for nasal diphtheria.

On examination the mucous membrane of the left nasal chamber appeared moderately congested with slight deviation of the septum at about its middle. The right nasal chamber was full of brownish, sticky crusts, on removal of which something hard and metallic was encountered. After cleaning nose by direct suction, the hard mass was cautiously removed. This was about the size of a pistachio nut, weighed 45 gr. and measured 1.95 cm. x 1.31 cm. x 0.65 cm., and

proved to be a stone. On probing, another hard mass was found to be lodged in the inferior meatus, manipulation of which caused great pain. The turbinates in the right side were practically absent, probably destroyed by pressure of the calculus. There was a mucopurulent discharge with papular pharyngitis. Roentgenogram showed a shadow on the right side of the nasal chamber. The next day breathing was entirely free on the right side, the hard mass disappeared, the patient did not remember swallowing or blowing it out. The mucous membrane was slightly congested and there was very little discharge. The patient never came back for further treatment.

Case 3: A. S., female, white, age 26 years. This Italian girl came to me on Aug. 31, 1932, complaining of pain along the bridge of the nose for the past five years, constant headaches, pain in the left eye while reading, clogged ears, postnasal discharge and subject to frequent colds.

Examination revealed a large brittle mass filling up the entire left nasal chamber, sharp above and below, somewhat mobile but painful on manipulation. On the right side the septum was markedly deflected above and below with deeply marked furrows between the deflections. Under local anesthesia a hard, brittle mass, the size of a half large-shelled walnut, which weighed 68 gr. and measured 3.98 cm. x 2.54 cm. x 1.2 cm. and had to be broken in two in order to remove it. Both the lateral and septal walls were indented and covered with granulations. There was a large posterior septal spur on the left side, touching the posterior part of the inferior turbinate, with a ridge extending forward. Following the removal of the stone, there was a profuse discharge for about a week and then cleared up completely the following week. All the symptoms the patient complained of completely disappeared.

COMMENT.

There are several points of interest in connection with the presentation of these three cases.

1. We are fortunate in having three principal varieties of nasal calculi; namely, Case 1 represents a false rhinolith because of a foreign body (the Indian nut shell) having formed the nucleus. Case 2 represents a true rhinolith, probably originated from a blood clot; its color being black. Case 3 also represents a true rhinolith, but its nucleus must have been mucopus as it is grayish white in color.

2. In Case 1 the nut shell must have been in the nose for many years (probably since childhood) without having given rise to any symptoms, as the patient does not remember having had a foreign body in the nose. It might have been forced into the nose through the posterior nares during vomiting, coughing or laughing while chewing Indian nuts.

3. Structural changes which should have resulted from the long sojourn in the nose were here prominent by their absence.

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391 Pennsylvania Avenue.

A CASE REPORT OF THROMBOSIS OF THE LATERAL SINUS, INFERIOR PETROSAL SINUS AND THE OPPOSITE LATERAL SINUS, WITH POSTMORTEM SPECIMEN.*

DR. JOHN MACKENZIE BROWN and DR. ROBERT J. BOWMAN,
Los Angeles.

E. L., a boy, aged 12 years, came into the office on March 24, 1931, and gave the following history: Both membrana tympani ruptured spontaneously during an attack of measles in 1921. The left ear had discharged for about three years and ceased discharging shortly after the tonsils and adenoids were removed in 1924. The right ear had discharged off and on ever since the attack of measles. On March 17, 1931, he had an upper respiratory infection with little or no fever until the 21st, when he had a chill and temperature of 104°. The chill and fever recurred on the 22nd and the 23rd. It was the following morning he came to my office.

Examination at this time revealed the left ear canal dry; there was a large central perforation in the posterior inferior quadrant of the membrana tympani, and he heard a whisper at fifteen feet. The right ear canal was filled with a foul thick discharge and the membrana tympani had a large marginal perforation extending above the posterior fold. The periosteum over the mastoid was thickened and the whole mastoid was very tender on pressure, hearing for loud voice only. There was no swelling in the neck on either side. The cervical glands were enlarged, more so on the right. The nose was subacutely inflamed with free breathing spaces on both sides. The sinuses were clear by transillumination, and the pharynx and larynx were negative. The tonsils and adenoids had been completely removed. Eye examination was negative.

A diagnosis of chronic mastoiditis with an acute exacerbation and probable lateral sinus phlebitis was made, and the patient sent to the St. Vincent's Hospital for X-ray, laboratory and physical examination and preparation for immediate operation.

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Examination in the hospital revealed:

Family and Past History: Essentially negative except for above.

Physical Examination of head, lungs, heart and circulation revealed no pathology.

There was a slight hyperactivity of the tendon reflexes.

The ears were as above.

Laboratory Findings: Urine: Reaction, acid; sp. gr., 1.010; albumin, 3 plus; sugar, 0; casts, many hyaline; W.B.C., 6-8 per HPF. Blood: Hb., 72 per cent; R.B.C., 4,200,000; W.B.C., 17,500; Lymph., 21 per cent; Polys., 78 per cent; Eosin., 1 per cent.

X-ray Report: Roentgenograms of the right mastoid area with similar views of the left for comparison showed the mastoid cells of both sides increased in density, more on the right than on the left, with no demonstrable cell walls present. Findings suggested a chronic right mastoiditis with considerable sclerosis of the bone.

Clinical Diagnosis: Chronic right mastoiditis with probable lateral sinus phlebitis.

Operation: The mastoid was very sclerotic. On going through the outer plate we found a large cavity full of cholesteatomatous material. When this debris was cleaned out we found the mastoid had been almost completely exenterated by the cholesteatoma. The plate over the sigmoid sinus for an area of 3 cm. had been destroyed and the sinus was very thick and dark colored. We then went below and exposed the jugular, which we found collapsed and the lumen obliterated. The vein was ligated. Returning to the mastoid we did a hasty radical, then exposed the lateral sinus up beyond the knee and down as far as we could. The sinus was opened and found completely thrombosed and partially broken down. We removed the thrombus as far down as we could go, but failed to get any bleeding. Posteriorly we had to go almost to the occipital protuberance before obtaining free bleeding. The sinus was left open throughout and packed with iodoform gauze down as close to the jugular bulb as possible and back to the torcular. A transfusion was given at this time. Culture from the mastoid showed a non-hemolytic streptococcus.

The patient ran an extremely high septic temperature with daily chills, the temperature ranging from 99° to 105°, and the last day

going to 107°. Three more transfusions were given, on March 28, April 3 and April 6. Acriviolet was administered intravenously on April 4. The packing in the sinus was changed on the third day and each subsequent day. Eye grounds remained negative. The boy died on April 8, 1931.

Autopsy (by Dr. E. M. Hall of the University of Southern California): The superior longitudinal sinus contains a tenacious, non-adherent post-mortem clot. The right lateral sinus is distended at the torcular end with a thrombus that appears as a solidified greenish yellow pus. The thrombus extends to the torcula herophili from which tags extend into the left lateral and superior longitudinal sinuses a short distance. The right inferior petrosal sinus is also thrombosed and partially broken down. The thrombus extends anteriorly to about the apex of the petrous portion of the temporal bone, where it is terminated abruptly. The jugular bulb is also thrombosed. The remainder of the left lateral sinus, the superior petrosal and the cavernous and circular sinuses are normal. The brain sectioned after hardening shows no focal lesion.

REMARKS.

Thrombosis of the inferior petrosal sinus is a very rarely diagnosed complication, not only of ear disease, but of any of the other diseases which so commonly cause a thrombosis of the lateral, circular, straight or cavernous sinuses.

Ballance,[1] Bircher,[2] Passow,[3] Poulsen,[4] Dwight,[5] Von Beyer,[6] Richards,[7] Holmes,[8] Hemmeon,[9] Kernan,[10] Muller,[11] Ballenger[12] and Shemeley[13] each report one or two cases.

Poulsen's case[4] presented a thrombosis of the superior petrosal sinus as well as that of the inferior petrosal.

The inferior petrosal sinus[14] begins in the posteroinferior part of the cavernous sinus, passing along the posterior inferior part of the petrous apex and through the anterior part of the jugular foramen, and ends in the superior bulb of the internal jugular vein. There is a valve at the jugular entrance of this sinus.

The exact way in which the inferior petrosal sinus becomes infected is questionable. Ballance[15] states that it may be by either (1) direct extension of the infection in the petrous pyramid to the sinus by direct continuity of tissues, or (2) by the way of a connecting sinus. This may be either downward in the direction of the blood

flow from an infected cavernous sinus or it may be, as in Passow's case,[3] against the blood flow from the bulb to the cavernous sinus through the inferior petrosal sinus. Ballance believes that septic venous infection can easily extend in a direction contrary to a blood stream so sluggish as that in the inferior petrosal sinus.[15]

Hemmeon[9] points out that the infection may pass through the petrosquamosal suture as well as directly through the bone to the sinus.

Thrombosis of the petrosal sinuses is generally an extension of the infective process through one of these channels; three cases are reported of a primary thrombosis in the superior and inferior petrosals.[3, 11, 13] In one case,[13] the diagnosis was made by Mackenzie before surgery and confirmed at necropsy.

In reference to the thrombus of the opposite lateral sinus, Page[17] quotes Richards in saying that a thrombus cannot extend from the opposite sides of the torcula as the rapid flow of blood through them at this point would tend to prevent the formation of the clot. Ballance[18] supports this theory and states that while this is found to be the general rule, it is not invariable. Cunningham[19] states that not uncommonly there is a communication between the right and left transverse sinus across the front of the internal occipital protuberance, and occasionally the superior sagittal, the two transverse, the straight and occipital sinuses unite anterior to the occipital protuberance in a common dilatation—the confluens sinuum.

In our case a thrombus actually did form in the opposite lateral sinus; however, we are of the opinion that it was a late manifestation and it was continuous from one lateral sinus to the other.

I believe the sequence of events in this case to have been as follows: First, infection of the sigmoid sinus, as the bone over the sinus was destroyed for a considerable area, the sinus walls were very thick and the thrombus had broken down in this area. This thrombus with the infection undoubtedly extended into the bulb and back into the inferior petrosal. The extension into the left lateral sinus was probably a late manifestation, as we had free bleeding from the torcular end at the operation. The fact that there was a complete obliteration of the jugular vein is strongly suggestive that the lesion in the sigmoid and bulb had been present for some time. Whether the new upper respiratory infection lighted up the old phlebitis or the old infection was smoldering and broke out at this time is problematical.

Ballance states that most infections of the venous sinuses travel in the direction of the blood stream, while Jones of Liverpool is of the opposite opinion. I believe the direction of extension is governed by the extent of the thrombus; if it is partial the direction will be down, and if the vein is completely thrombosed it will be backward. I believe that phlebitis of either petrosal is most frequently due to infection of the petrous pyramid in the course of an otitis media or mastoiditis, and the balance of these petrosal infections mostly come from extension from the jugular bulb.

The symptoms that would lead one to suspect involvement of the petrosals are: first, pain over the course of the fifth nerve; second, sepsis manifested by chills and fever in the presence of an acute or chronic middle ear disease, or third, in the course of a suspected sinus phlebitis after you have ligated the jugular, satisfied yourself that there is no pathology in the sigmoid or beyond and have irrigated the jugular bulb, the septic symptoms continue. I believe one is then justified in thinking of the possible involvement of the petrosals.

The treatment consists in direct attack on the petrosals through the jugular bulb for the inferior and by removal of the labyrinth as suggested by G. W. Mackenzie[13] for the superior.

Although very few cases of thrombosis of the petrosal sinuses have been reported, it is probably a much more common condition than we now believe it to be. It is usually secondary and its symptoms are masked by those of its common precursors, namely, petrous apex lesions, lateral sinus thrombosis and cavernous sinus thrombosis. Its treatment is drainage after internal jugular ligation and whole blood transfusions. Dr. George Coates[21] recommends immunized donors.

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1136 West 6th Street.

SINUSITIS IN CHILDREN — FROM THE RHINOLOGICAL VIEWPOINT.*

DR. HERMAN B. COHEN, Philadelphia.

In the time allotted it is quite impossible, nor is it essential at this session to go into any detailed anatomy of the sinuses, however important this phase of the subject is. A brief resume will be beneficial with the view of making my remarks as practical as possible.

All sinuses, we are told by such an authority as Schaeffer, begin their development at about the hundredth day of fetal life and the entire series probably communicated with each other originally. This is a very important fundamental fact and helps us in the understanding of the postnasal development. Also important, is the study and attention given the individual sinuses and to recognize and treat abnormally developed sinuses, such as a small or absent frontal, a double maxillary or misplaced ethmoid cells. I must hesitate here lest I get too deeply involved and hasten to present what we have to deal with in life. So then, *at birth*, as indicated on the chart, we have the maxillary or antrum of Highmore as present, though small as a bean, on the nasal side of the orbit, reaching full development at from 15 to 18 years of age. The ethmoid sinus or ethmoid labyrinth, which is a better term, is the only accessory nasal sinus always present at birth quite in detail, and divided into anterior and posterior groups of cells. The frontal is absent, arising or developing as it will later from the ethmoid labyrinth. The sphenoid sinus manifests itself only as a faint depression.

At one year of age the frontal is still absent, but the sphenoid is a distinct cavity reaching full development at about seven years. At three years of age the frontal makes its appearance and is only a distinct cavity at about seven to nine years of age. It is apparent, then, that the frontal is the tardiest of them all. All this is of practical importance and we must not be unmindful of the anatomical variability—either a precocious or a tardy development of the sinuses.

We arbitrarily say that infancy is the first two years of life; childhood, three to 12 or 13, and as far as the subject of the evening

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is concerned, this is the period of life we are to deal with. An affection of the ethmoid labyrinth would, therefore, for all practical purposes, be the only set of sinuses to be concerned with in infants. The maxillary up to two years of age is still quite small, although a distinct cavity, and can be treated as you would the ethmoid group at this age. A child of three to seven or eight under our care presents a larger group of sinuses for investigation, namely the ethmoid, maxillary and sphenoid, and, in that order of incidence of infection. About the age of nine or later, of course, the frontal may be involved and should be considered.

CHART I.

Sinus Development — Postembryonic.

	At Birth	*One Year*	*1-3 Years*	*7-Years*	*7-9 Years*	*15-18 Years*
Maxillary (Antrum)	Present (Small bean)	.....	.....	.....	.....	Fully developed
Ethmoid	9-14 cells (Curran)	.....	.....	.....	.....	Fully developed
Frontal	Absent	.....	Appearance	Pea-size	Distinct cavity	Fully developed
Sphenoid	Present as a rule	Distinct cavity	.....	Full development	.....	.....

ETIOLOGY AND GENERAL CONSIDERATIONS.

Etiologic factors should not be hard to discover, as in children the condition of the sinuses, though it may be chronic, cannot be of many years standing.

Intranasal abnormalities are predisposing causes. So are poor hygiene, improper clothing and improper heating and ventilation of our homes. Nephrosis and allergy by producing a serum logged mucous membrane are important factors. It is well to remember that nasal allergy may exist with negative cutaneous reactions. We know, however, that the end result of the etiologic factors is a mucous membrane altered in structure and becomes a favorable soil for bacterial invasion. (This may be the beginning of a vicious cycle for allergias.) The pathology will vary from moderate to a permanent change and be either hyperplastic or atrophic, or both. Depending upon the virulence, the infection will be mild or severe, and will also depend upon the size and contour of natural ostia or canals. Small ostia with swollen membrane will spell empyema and pressure may cause rupture to or through other structures.

Obstructive adenoids cause retention of nasal secretion. The latter causes irritation with resulting inflammatory changes. Through con-

tinuity of structure we have produced a general catarrhal condition. Obstructive causes impair natural sinus drainage or cleansing; i.e., natural blowing, sniffing and hawking. Exanthemata, especially measles, scarlet fever and diphtheria, are often important etiological factors, and so are also swimming and diving.

Systemic Factors: Diet deficient in certain vitamins, especially A and D; allergia and endocrine imbalance are to be seriously considered. There remains much to be done in experimental investigation regarding the underlying causes, including deficiency in calcium, the relation of the disease to metabolic disorders, changes in the blood chemistry from the normal, etc. How about heredity? Frequently we see children of parents suffering from sinus disease, contract the disease early in life and irrespective of environment, diet or possible contagion. An inherited weakness toward the condition, be it as a result of endocrine imbalance or whatnot, is an important factor worth considering.

Does the removal of tonsils and adenoids in young children predispose them or cause them to succumb to sinus infection? I do not believe so. Where there are definite indications for adenoid or tonsil removal at no matter what age, it should be done. I can only see helpfulness for the child. When you examine a child following a tonsil and adenoid operation and if glands persist or especially if the so-called tonsil gland (the one lying beneath the posterior belly of the digastric muscle) becomes enlarged, investigate the sinuses. Why? On account of the lymphatic drainage of the sinuses to that gland and posterior cervicals.

Symptoms: Subjectively the symptoms will naturally depend on the age, and as children are often unreliable in their statements, we should rather look for signs.

1. Open mouth breathing.

2. Discharging nose—especially intermittent discharge and unilateral.

3. Alar or nostril irritations.

4. Cough (very significant).

5. Asthma or asthmatic breathing.

6. Eye signs: Conjunctivitis. Lid swelling, especially the upper. Keratitis, iritis. Optic nerve involvement, etc.

7. Gastro-intestinal disturbances, the result of swallowing purulent and putrefactive material.

8. Pharyngitis—acute or chronic. We frequently see the granular type.

9. Bone involvement, especially from suppurative tooth sockets and roots. The ethmoid and frontal sinuses are the commoner ones to present bone involvement, often presenting fistula formation.

10. Recurring fever.

11. Aprosexia (the inability to fix the attention).

Diagnostic Aids and Signs:

1. X-ray: Because of the age, the absence of former causes predisposing to a thickened membrane, X-ray becomes more valuable than in adults.

2. Suction: Mild, two to three lbs. of negative pressure for mass suction.

3. Transillumination: This test is of doubtful value in children; because of haziness in cases of thickened membrane, thickened bone, absence of a frontal sinus, and in children where the tooth buds of the permanent teeth are under part of the antrum. This test is also of doubtful value in cases where there has been an old process in the frontal or antrum of older children, which healed. X-rays, well made, will show the amount and character of sinus involvement.

4. Pharyngoscopic examination: Is helpful in the diagnosis of sinus infection, but cannot always be used in children.

5. Bacterial and cytological study of the antrum through needle puncture.

6. Repeated colds in a child, especially one that is not thriving and is anemic, should put us on our guard.

7. An irritable and cross child.

8. Headache: If the child is old enough to complain, is an important symptom. Its absence means nothing.

9. Adenitis.

10. Nausea and vomiting and possible convulsions are often symptomatic of sinus infection.

Complications: As a focus of infection, complications will clear up if the sinuses are treated properly and drained. The possible complications are: *1.* meningitis; *2.* brain abscess; *3.* optic neuritis;

4. cavernous sinus thrombosis; *5.* albuminuria; *6.* arthritis; *7.* cardiac disease. The commoner complications met with are: *1.* bronchitis (sinus chest); *2.* gastro-intestinal disturbances; *3.* conjunctival inflammation; *4.* middle ear and mastoid infection; *5.* orbital cellulitis and abscess; *6.* pyelitis; *7.* atrophic rhinitis.

Prognosis: A thorough study of the underlying causes will save many a patient from unnecessary surgical meddling. Thus, a properly cared for child with sinus infection; the none too hasty surgery in the acute stage, except where danger signs appear, will bring about prompt and satisfactory results.

Treatment: Treatment by rhinological methods alone is a most unsatisfactory procedure, particularly for the chronic type. The child may get well or improve, but will soon return on account of another cold with the same systemic disturbance. Associated treatment by a pediatrist will bring home to us the fact that "these cases are being investigated as patients before they are investigated as cases of sinusitis."

1. From the rhinological point of view: drainage (sinus and nasal).

2. From the general medical point of view: Metabolic, general medical care, etc. This phase I will not touch upon.

Rhinologically: Relieve nasal obstruction; remove the adenoids and possibly the tonsils at the proper time.

Cleanliness of the nose by catheter and mild negative or mass suction. Here, the production of Bier's hyperemia is an important adjunct. Shrinkage with the use of weak cocaine with or without adrenalin or ephedrine. Rest in bed for all fever patients. Vaccines—or non-specific protein injection as used by Mithoefer of Cincinnati. Fresh air and sunshine. Limit the amount and correction in the type of swimming.

For the Chronic Cases: Only after thorough and careful conservative treatment fails and when patient presents serious signs and symptoms should surgery of a radical nature be considered. For the frontal: Remembering the developmental anatomy, one must be careful and avoid intranasal frontal operation under twelve. For the ethmoid: If partial turbinectomy has not proven of sufficient help, external operation is the safest, though rarely necessary in the uncomplicated case. The sphenoid may be opened at the same time if there is any question of involvement.

Antrum: Where the permanent teeth have erupted the operation is simple. Radical operation should only be done after repeated needle punctures in an older child; where a self-retaining catheter in a younger child, or a window operation have failed.

Given then a child, 3 to 9 years of age, with sinus infection in a subacute stage, what would be our treatment?

1. Hospitalization if at all possible.

2. Complete and thorough history and study from every angle as mentioned under etiologic factors. This is the "sine qua non" for the successful treatment.

3. Drainage: Spray the nose t.i.d. or oftener as the case demands with an aqueous cocaine sol. one-half to 1 per cent with ephedrine 1 per cent. Antipyrine may be added to prolong the action.

Gentle mass suction of 2 to 3 pounds followed by aspiration with catheter.

Careful irrigation with warm normal saline alone or with bicarbonate of soda.

Tamponage with 10 to 20 per cent silvol or argyrol, properly placed in the spheno-ethmoidal recess and under the middle turbinate if possible. These are left in for 20 to 45 minutes, the patient sitting or lying under an infra-red lamp. In obstreporous or recalcitrant children or where for some other reason tampons cannot be used, the child is laid upon its stomach, the head hanging over the edge of the table or sofa, 15 to 20 per cent silvol drops (about 10) are instilled.

A second irrigation of warm solution is followed by a soothing sedative oil spray.

Exposure of the face and body to the quartz light is very helpful and is used by many of us.

Medicated steam is often recommended and is used very often by the writer.

At the end of four to six weeks, with little or no improvement, the X-ray having been negative for antrum involvement, partial turbinectomy is done where there is any reason to believe they are obstructing the ostia. The antra are punctured, sterile salt solution is installed, withdrawn, studied and kept for vaccine if wanted.

If the X-ray report was positive for maxillary infection, then, of course, puncture and irrigation is done earlier, and depending upon

the findings whether a self-retaining rubber catheter is left in situ or a window made inframeatally or in the pars membranacea, age being the deciding factor for the latter.

Since the rhinologic history and findings, including X-ray plates in allergic individuals are so similar to those seen in chronic infectious sinusitis, it is of first importance that we differentiate the two conditions and therefore no case of chronic sinus infection should be put up for operation before having been studied for allergic reactions.

What about the child with an apron of lymphoid tissue in the pharynx and epipharynx? Yes it has had the tonsils and adenoids out once, maybe twice. Get after the sinuses and have the patient exposed to X-ray. If old enough, try electrical destruction, but most important of all is thorough study as to cause, including allergy and blood work.

SUMMARY.

1. The age of the child to be treated is very important in the study of the specific sinus involved.

2. Sinusitis in children is a distinct entity underlying not only nasal symptoms, but causing disturbances in distant as well as adjacent organs or structures.

3. Etiologically, sinusitis in children is a complex problem. Therefore, a thorough study of the underlying physiologic and biochemic dysfunctions in the individual patient.

4. Allergy plays an important role.

5. Only by co-operation with the pediatrician and by study and investigation with every clinical and laboratory facility at hand can the rhinologist be of service in these cases and get satisfactory results.

6. Thoroughness being the keynote, many cases will be cured or so improved that surgery will be unnecessary.

13th and Spruce Streets.

FRONTAL OSTEOMA; MOULDING MISPLACED FRONTAL SEPTUM; CASE REPORT.*

DR. Z. WILLIAM COLSON, Lawrence, Mass.

The only excuse for this report is the unusual anatomical relationship of the osteoma and also, possibly, the symptoms it produced.

Mr. E. F. M., white, age 38 years, a butcher, was referred by Dr. Harold R. Kurth, July 5, 1932, with a diagnosis of bony growth in right frontal sinus. The patient brought with him a skull X-ray taken by Dr. Kurth.

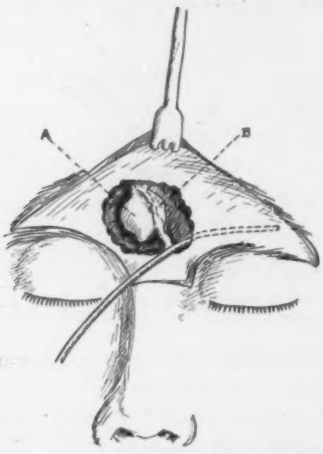

Fig. 1. (A) True frontal osteoma attached to anterior frontal wall. (B) Rounded misplaced frontal septum moulded by osteoma. Probe shown in left frontal sinus.

Chief Complaint: Dizziness, occasionally accompanied by nausea and vomiting; right frontal pain and "fluttering of nose."

Present Illness: The present illness began two years and five months before my examination, with an attack of dizziness which caused only one day of disability. Further attacks of dizziness have

*Drawings by Dr. R. J. Frackleton, of Massachusetts Eye and Ear Infirmary.

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recurred intermittently during the past two years, and have not responded to various forms of treatment. During these attacks he has fallen occasionally to the floor, and he believes that he always falls to the right. Extreme nausea and vomiting have accompanied a few attacks, and hematemesis was present once.

In March, 1932, the patient was referred to Dr. Ayer, of Boston, who was unable to find any organic nerve lesion to explain the symptoms, and made a diagnosis of psychoneurosis.

The dizziness is present most of the time, and ten days ago he had a severe attack and had to be carried to bed, where he remained for two days. When the vertigo is at its worst he has a dull pain in the right frontal region with a sensation of severe pressure above his right eye and a feeling in the right nostril as though it were "flapping in and out." On close questioning, this sensation of flut-

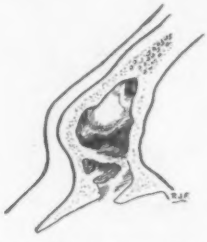

Fig. 2. Medial view of frontal septum showing convexity caused by pressure of osteoma.

tering in the right nostril is found to be synchronous with the pulse beat.

Physical Examination: Slight hard bulging of skull in right frontal region. Positive Ewing sign on right. Eyes: No nystagmus in any position. Discs show no choking. Nose: Marked irregular deviation of septum to left, only moderately obstructive. Transillumination: Small dull area on anterior frontal wall, about ¾ inch to the right of mid line. Both antra clear. X-ray (Read by Dr. A. S. McMillan, of Boston): Right frontal osteoma attached to anterior frontal wall. Wassermann: Negative.

Operation: July 13, 1932, at Lawrence General Hospital. Spectacle incision to periosteum under both eyebrows and across bridge of nose. Periosteum incised above right orbital edge and wide retraction upward over right frontal area. Entrance was made through the anterior frontal wall just medial to the point of maxi-

mum bony bulging. A large rounded bony mass was disclosed just lateral to the window of entrance. This mass was almost twice as large as the osteoma shown by X-ray. A bent probe could not be passed around it. An opening was then made lateral to the mass, and attempts to pass a probe from that side were likewise unsuccessful. The chisel was then applied above and below, and the bony mass was removed in toto, along with a portion of the posterior frontal wall, exposing intact dura. It was then discovered that the rounded bony mass seen through the first opening was the misplaced frontal septum, moulded like a walnut shell around the osteoma. Incision closed with small rubber dam drain at juncture of nose and right eyebrow.

Fig. 3. Sketch made directly from X-ray showing true position of osteoma and septum.

Pathological Report: Osteoma of frontal sinus.

Postoperative Report: Aug. 30, 1932, Dr. Kurth sends the following report: "Since his operation for removal of osteoma of frontal sinus, patient feels most optimistic and states that he feels freer of his symptom complex than he has been at any time during the past two years. He still has, at times, on becoming over-tired and fatigued, a certain sensation of dizziness, but during the past week especially, he has been free of any symptoms whatsoever."

Dr. Kurth's report, Sept. 30, 1932: "Patient came in to see me yesterday and states that since his operation he has put on ten pounds weight, has lost the symptom complex of headache and dizziness entirely, and seems to be in a much more optimistic state of mind with regard to future relief of the symptoms which he has been troubled with for the past two years. He feels that he will be entirely free of the symptom complex."

253 Essex Street.

ARGYRIA IN A CHILD FOLLOWING INTRANASAL USE OF ARGYROL.*

Dr. Myron A. Zacks, Philadelphia.

Synonyms: Argyria, Argyrism, Argyrosis.

The unusual occurrence of this pigmentation in a child following repeated and prolonged use of argyrol in the nose, presenting interesting and unusual findings apparently not mentioned in the literature, prompted the review of this subject. From an otolaryngological point of view, few, if any, medicaments have gained the widespread popularity and use as the various organic silver preparations, particularly argyrol.

Argyria may be defined as a slate gray or bluish discoloration of the skin, deeper tissues and organs due chiefly to the deposit of insoluble albuminate of silver which results from local contact and ingestion of a soluble silver salt.

It is of historic interest to know that the therapeutic use of silver salts, the oxide, and later the nitrate, dates back to the Arabs. Its benefits were in some mysterious fashion attributable to astrological influences, and it was the belief during that period that nervous diseases were markedly affected by phases of the moon which were unexplainably linked with silver in the system (hence lunar caustic, lunacy). Incredible as it may seem, the treatment of insanity even as late as the eighteenth century with silver nitrate was based and practiced on this fantastic teaching.

At present, silver poisoning is comparatively rare, although it was not an uncommon occurrence during the first half of the nineteenth century, during which period silver nitrate was extensively used in the treatment of epilepsy and tabes.

The first case of argyria was reported by Angelus Sala, in 1647, from the internal use of silver nitrate in the treatment of epilepsy. However, it was Frommann's classical paper in 1859, detailing case history, autopsy and histological findings that gave the medical world

*Read (Case Presentation) before Philadelphia Laryngological Society, Jan. 3, 1933.

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its first scientific understanding and appreciation of this clinical entity. Subsequently, from time to time, various articles and case reports appeared in the literature, until at the present time we have a fairly clear concept of this interesting pigmentation.

Argyria occurs in two forms: *1.* Widespread or generalized. *2.* Localized.

The widespread or generalized, with few exceptions, follows internal medication, utilizing as carriers the lymph and blood stream.

Localized follows regional medicinal application and is also observed in silver metal workers (occupational argyria). In this form the silver penetrates directly the area of contact (skin or mucosa) and the discoloration never goes beyond this limited site.

Myers, in a careful review of the literature, records 36 cases of localized argyria since 1857 and 44 cases of generalized since the first case reported by Sala in 1647. Based on these figures, it will be noted that one form is about as prevalent as the other. Of the forty-four generalized cases, five were due to nose and throat application of silver nitrate, and one of these, reported by Goldstein in 1921, was due to "throat paintings" with argyrol twice daily for a period of one year. Of the thirty-six localized cases, two were due to silver nitrate application to the rhinooropharynx.

In the remaining 73 cases the generalized form was due mainly to the ingestion of silver nitrate for gastrointestinal disorders and lues, while the local form was due principally to conjunctival and urethral application and industrial exposure.

Many theories have been advanced as to the modus operandi concerning the chemical aspect of this pigmentation. However, the most logical probable explanation is as follows: When silver is given for prolonged periods, probably only a minute fraction is absorbed as a silver organic compound, probably the albuminate. Unna labeled the combination elastin silver when it affected the elastic tissue fibers and collogen silver when the collogenous tissues were involved.

The histopathological changes caused by the silver compound consist of a deposition of the pigment in the elastic tissue fibers of the body, especially the subpapillary layer of the skin and mucosa. However, in the more advanced there is an occasional tendency for the epidermis and connective tissue cells themselves to absorb and deposit this pigment. The amount required to produce this pigmentation varies from 15 grains of silver nitrate to as much as 400

grams of silver. The time varies from two and a half months to twenty years.

In making a diagnosis careful case history reveals the administration or application of any of the silver preparations over a variable period of time, followed by slate gray or bluish discoloration of the patient, either of the generalized or localized type. Mention in literature is made of gastrointestinal disturbances, blue line on the gums, anemia and nephritis caused by this silver poisoning.

In differentiating this condition from those which bear resemblance to it, the following must be borne in mind: Hemochromatosis, Ochronosis and Addison's Disease.

In the generalized form, the staining is more or less permanent. In the localized form, however, it may diminish or even disappear spontaneously.

The treatment consists of the permanent discontinuance of the use of silver in any form. Urotropin by mouth has been tried for purposes of decolorization with encouraging results mentioned. It is probably best to avoid prolonged and undue exposure to strong sunlight or ultra violet lamp rays.

Case History: Oct. 8, 1930, E. C., female, age 10½ years, was referred, suffering from severe headaches, restlessness, inability to concentrate and general physical deterioration. Onset of symptoms about four years ago and in spite of the fact that she has been under specialists (otolaryngologists) care since the onset, symptoms were becoming more severe. Up to the time patient was referred, each otolaryngologist prescribed argyrol solution (exact strength not known by patient), which prescription patient herself renewed from time to time, using this medication in the form of nose drops two or three times daily.

Examination—Nose: Mucous membrane fairly normal in appearance. Both nasal chambers, particularly the left, filled with thick mucopus. Limited amount of intranasal breathing space owing to somewhat boggy inferior turbinates. Septum is deviated high on right and along the suture line, almost in contact with the inferior turbinate. Both middle turbinates are somewhat wedged between the septum and the lateral nasal wall.

Mouth: Lips dry, mucosa of normal color. Teeth and gums negative. Pharynx is red. Lymphoid masses present in both fossa, posterior pharyngeal wall and extending upward into the nasopharynx.

Ears: Both drums are intact, somewhat retracted, color normal, with some scarring on the left side.

Transillumination: Both antra somewhat indistinct. Frontals (if any) are small and fairly clear.

Diagnosis: Suppurative Ethmoiditis (Bilateral). After a week of treatment, the symptoms showed little if any tendency to abate and it was deemed advisable to refer the patient to the Mt. Sinai Hospital (Nov. 14, 1930) for further detailed study.

Nov. 14, 1930, X-ray by Dr. George Rosenbaum: "The frontals are small, asymmetrical and clear. Sphenoids are clear. Both ethmoids are slightly clouded. Maxillary sinuses are large, symmetrical and clear."

Nov. 16, 1930, under general anesthesia the writer removed the anterior portion of the left middle turbinate, infracted the right middle turbinate toward the mid-line, removed lymphoid tissue from both fossa and nasooropharynx. Both antra were lavaged and found free from content.

Nov. 18, 1930, culture from nose for autogenous vaccine showed the presence of Friedlander's bacillus and staphlococcus albus.

Patient was discharged from the hospital after a fifteen day stay, considerably improved. The patient continued to see the writer and except for an occasional attack of headache, progressed generally favorably.

Oct. 23, 1931, examination of left ear revealed an acute suppurative left ear.

Oct. 31, 1931, slight discharge from left ear. Examination of right ear revealed bluish black discoloration of the drum throughout its entirety. Distinct anterior and posterior folds, particularly the posterior. Drum is retracted. The malleus handle is clear throughout its length. Cone of light is present. Posterior half of drum is fairly equally divided by a slightly elevated horizontal fold which passes from the umbo slightly downward and backward. When seen through the otoscope the vibration of the posterior half of the membrane above and below the fold is fairly good. On inflation with Politzer Bag, the posterior superior portion of the drum bulges markedly and assumes the appearance of an opalescent bleb retaining this appearance for quite some time.

Nov. 21, 1931, discoloration is less marked on the right. Bluish discoloration is now present in the left.

Dec. 6, 1931, the appearance of the right drum is fairly normal, but the left still shows evidence of the bluish discoloration.

March 5, 1932, patient given short Alpine Sun Lamp (mercury) exposure to face and neck. During this exposure mother observed for the first time a bluish black discoloration of the mid portion of the face and called the writer's secretary's attention to this observation, and which was readily confirmed.

March 10, 1932, mother states that members of patient's family commented on the bluish tinge of the skin of the face. Examination at this time revealed slaty discoloration of the skin of nose, inner portion of both lower lids, upper lip and especially the nasolibial fold area on both sides. Mucosa lining of the nose, especially the outer wall, cheeks, gums and pharynx, showed unmistakable slate blue discoloration. Conjunctiva clear.

Ears: Right drum is intact, retracted. There is a slate blue discoloration of the entire drum more marked along the periphery in the lower half. In the posterior inferior quadrant there is a black, irregularly round, firmly imbedded, discolored mass about 2 mms. in diameter. Left drum: dry perforation in posterior superior quadrant. Color is slightly slate blue, but less marked than the right.

Dec. 14, 1932, general physical examination by Dr. Maurice Vaisberg essentially negative. In addition to the above findings, the pigmentation was noted in the skin of the neck and finger tips.

During the time that this patient was under the writer's care she continued the use of argyrol, particularly when "colds" were threatening or actually present. This was done with the full knowledge of the writer, who took no steps toward its limitation or control in view of the fact that the patient volunteered the information that this drug not only gave her relief, but did not cause any discomfort during or following its use. However, when the first symptom of argyria manifested itself the argyrol was immediately withdrawn and the patient warned against the further use of any silver preparation. Thus it will be noted that the patient, at present only twelve years of age, used argyrol drops in her nose without any warning or caution as to its possible ill effect for a period of six years and suddenly developed argyria, exhibiting its first manifestation while under the mercury lamp.

A few days later this discoloration was sufficiently obvious to cause comment by various members of patient's family.

COMMENTS.

1. A permanent discoloration of skin and mucous membrane may result from the local use of argyrol. Hence, once stained, probably always stained.

2. This complication rarely occurs in the practice of an otolaryngologist.

3. This case differs from the type of generalized argyria usually described in that the areas involved are limited to the head, neck and finger tips.

4. It is fair to assume that the argyria resulted from the swallowed argyrol rather than from the amount absorbed from the local area.

5. There was absence in this case of any definite gum line discoloration and gastrointestinal disturbances.

6. The appearance of the ear drums is most interesting and unique and insofar as the writer is aware, is the first mention of this finding in the literature. This finding resembles closely a finding in a case reported and presented at this society by the writer in 1924, under the caption of "Hemosiderosis of Tympanic Membrane," and which as the name implies, is due to a deposit of hemosiderin in the drum.

7. Insofar as the writer is aware, this is the only recorded case presenting a sudden appearance following ultra violet lamp exposure.

8. The unsightly discoloration may present a medico-legal aspect.

9. With the establishment of the diagnosis it seems advisable to withhold any ultra violet exposure and to push the use of Urotropin.

10. A note of warning must be sounded against the unbridled and prolonged use of argyrol in rhinolaryngological practice. It behooves the practitioner in every case where he employs these medicaments to make inquiry as to the previous prescription and use of silver preparations and bear in mind the possible occurrence of this unfortunate and lasting complication.

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1633 Spruce Street.

THE LABORATORY IN LARYNGOLOGY.

Dr. Herman Friend, New York.

When one considers the tremendous advances made, and the great importance that has been attached to the laboratory, particularly in the last few years, it is really surprising how pathological studies have been so sadly neglected in laryngology. While it might be said that general medicine is already burdened with too many tests, many of which are of relative unimportance, it is equally true on the other hand that there are many examinations which would be of infinite help to the laryngologist, but for no good reason are simply not done. From a clinical standpoint, it has been said, "That by far too many and too few tonsils are removed." The truthfulness of this statement is borne out when one considers the large number of cases that are not benefited by tonsillectomy.

Bacteriologically, it is well known that the tonsils are an excellent storage plant in which any variety of organisms, namely, note the staphylococcus, streptococcus, micrococcus catarrhalis, colon bacillus, tubercle bacillus and many others, harbor themselves; therefore, cultures need not necessarily be taken to confirm that which is clinically obvious, but should rather be directed toward the possible diagnosis of other perhaps serious conditions.

I have, in examining many Gram stains, taken from growths on various types of agars, noticed an encapsulated Gram negative bacillus. Ziehl-Neelsen stains of the same growths showed an acid-fast, encapsulated bacillus which, morphologically was and at other times was not exactly typical of what we recognize as Koch-Weeks' organism. I have also noticed what appears to be an acid-fast streptococcus, or what might be a nodulated bacillus having chain formation, that is, end to end, which so closely resembles a streptococcus as to perhaps be mistaken for one by the inexperienced worker. Clinically, all these cases had a cough and had been previously diagnosed as bronchitis, asthma

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or bronchial asthma. In some cases X-ray examination revealed nothing, or at times a very slight suspected area. Sputum examination would at times not differ very much from the tonsil findings and at other times was definitely confirmatory. I have also frequently found this peculiar acid-fast bacillus in sputum, and while I did not feel justified in calling it a tubercle bacillus, I felt skeptical enough to make the finding known to the clinician, in which case former diagnosis was frequently changed to tracheitis, bronchial tuberculous asthma or tuberculosis as clinical and laboratory findings warranted. It is interesting that the French report the recognition of some six types of acid-fast bacilli and streptococci, one of which they name a streptobacillus, and they further lay claim to having found tuberculosis of the lungs on postmortem in all these cases. The Germans not only substantiate this finding, but also lay claim to having found tubercle bacilli in tonsils.

In tuberculosis, the acute and chronic stages are quite obvious to the clinician, but I know of few conditions that remain so absolutely undetected as the incipient stage, even by good clinicians. From the laboratory standpoint, the examination is most difficult and time consuming, but as in many other conditions, a fairly careful examination of the slide in general will give the experienced laboratory worker a cue, for there is somewhat of a general picture of what one might expect. It might be well to add here that a ten or fifteen minutes search, unless by chance the organism is found at once, is useless; properly done, every part of the slide must be carefully searched, which takes hours and, in some cases, it might be necessary to examine many slides, bearing in mind that one organism is diagnostic. I have also found upon culturing tonsils, what I believed to be a tubercle bacillus and have frequently confirmed this with sputum examination; X-ray at times showed lesions in the lungs, and at other times family history bore out this evidence. It was also gratifying as well as very unfortunate, on three occasions I found tubercle bacilli in both sputum and tonsil cultures against three negative reports of others and in each case the patient died following hemoptysis, and in one case hemorrhage. I might say without fear of being imprudent that in the incipient stage the laboratory is perhaps the only means for diagnosis, apart perhaps from a most careful and thorough application of all clinical methods. I do, however, wish to stress tonsil examinations for

tubercle bacilli, for in my experience I have detected an acid-fast encapsulated bacillus in many a tonsil before the patient complained and, incidentally, before there were any clinical signs, for when the condition has progressed, undetected, it is frequently too late. In talking to a nose and throat man recently, I was informed that there is no reason why this organism would not find a very suitable media in tonsillar tissue. Unfortunately, no one has taken the trouble to section "apparently healthy" tonsils, hence we do not know whether the very crypts would show any lesions.

There is one place in laryngology where the use of the laboratory cannot be overemphasized and the one place where the clinician is frequently disappointed, as well as puzzled, because of the absence of the clinical picture (lesions)—the acute throats. Last summer I saw one case, and recently a doctor asked me to see his own son. In both cases the children were very sick, high fever, toxicity, and suffering from extremely sore throats. Clinically, the tonsils were enlarged and very markedly inflamed, but no membrane was present in either case, in fact nothing that would lead to a diagnosis was present. Gram stain showed Vincent's angina to be absent. Cultures in both cases showed pure growths of bacillus diphtheria. In the latter case, a typical membrane formed two days after the laboratory diagnosis and one day after the administration of antitoxin. I also saw a case two months ago that was very striking from many angles. The patient was but eight months old and had been sick for about a week; the doctor in attendance refused to consider it diphtheria only because the infant was still within the age where immunity against infectious and contagious disease is a commonly recognized and accepted standard. A colleague, however, was consulted and the latter called me in; clinically, the throat was perfectly clear, but there was a definite lesion on the dorsal end of the tongue, which in all but its site was absolutely typical of diphtheria. Cultures taken by me showed a pure growth of bacillus diphtheria in eight hours; antitoxin administered cleared up the case in three days. I have seen many similar cases, some with atypical lesions which proved on culture to be staphylococcus infections, and I remember three cases quite distinctly where no lesion at all was present, that turned out to be Vincent's infection. These cases, let me repeat, did not have the slightest clinical evidence (no lesions), other than high fever

and suffered from extremely sore throats. The positive slides were further proved to be correct, by rapid recovery under specific treatment, and it must be borne in mind that Vincent's must be treated specifically, if it is Vincent's. It is not unusual for both diphtheria and Vincent's infections to lack the clinical lesions at the onset of the infection, hence if the condition can be caught early by the patient's indisposition and a laboratory diagnosis, both can be quickly aborted before anything serious develops. It is hardly necessary to mention that diphtheria and Vincent's may occur together, and I have seen two cases of Vincent's and tuberculosis of the throat, that is, where the Vincent's sore throat led to the finding of the other. There is also a type of throat and a condition of the teeth which dentists insist upon calling by the pet name of chronic Vincent's. Clinically, the first, or medical, throat very closely resembles Vincent's, but a Gram stain shows in the first place a complete lack of the usual flora which in Vincent's is typical; fusiform bacilli are absent, and while spirochaeta are present, they will be found to be thicker, longer and contain less spirals. Whatever this organism is, it is not Vincent's. These cases clear up in twenty-four hours. Regarding the dental chronic Vincent's, I have observed but two or three slides of this supposed organism, which I am led to believe might be a fungus. The finding of spirochaeta does not establish diagnosis until the organism has been identified, as there are many kinds of spirochaeta, as the causative organisms in various infections.

In regard to tonsillectomy, while I say without reservation that I am yet to see a healthy pair of adult tonsils, when or how do we know that the tonsils are the cause of systemic disturbances? Other than the clinical evidences, such as *inflammed pillars* (tonsils may be enlarged or buried), fatigue, hypotension, secondary anemia, and at times arthritis, etc., which might have the cause elsewhere or perhaps in combination with diseased tonsils, the blood count may be said to be an absolute guide; in fact, it is this very "tonsil count" that often results in perplexities in other acute conditions, where the usual laboratory picture is not obtained. The blood stream being in intimate contact with and acting as a communication between the various organs, is by virtue of this function an excellent and very sensitive index of the state of health of the human organism. While it might seem perfectly absurd to even mention the complexity of this

human organism, yet, strangely enough, this very complexity relative to the blood count, the index, has been sadly neglected, and is the very reason why in many cases the classic picture is not obtained. We have not been mindful of the possibility of two or more existing conditions in apparently healthy persons, each of which conditions may affect the blood stream, giving rise to either similar or extremely opposite pictures, thus offsetting the expected one, hence the cause of much disappointment.

We all know that any acute, nonlymphatic, inflammatory process with resistance, generally gives rise to a leukocytosis with a high polymorphonuclear cell count. Any textbook on the subject informs us of a multitude of clinical entities, each of which may be identified by its classic picture. However, while some observers have reported it, it is not generally known that there is a definite tonsil blood count and one that causes much confusion. Infected tonsils, but only when they affect the system, alter the blood picture in a given way. Infected tonsils that affect the system always give rise to a leukopenia, a lymphocytosis (small cells) and a secondary anemia. Diseased tonsils being a most common thing, I have in doing routine blood counts as part of a general examination seen the two in thousands of cases. I have also had the fortunate opportunity in many cases of repeating the blood count postoperatively, and observed a definite change in the blood picture at times within several hours or simultaneously with the disappearance of the patient's complaint. In order to avoid confusion, in regard to other conditions where a leukopenia is the rule, such as typhoid fever for example, the differential count, together with the clinical findings and possibly other tests, will clear this point. The blood count then should be the surgeon's guide as to what he may expect in regard to the patient's complaint, or whether perhaps the cause may be elsewhere, or may be a combination of conditions, in spite of bad tonsils being present. I am not foolish enough to even think that this will go unchallenged, for I have known surgeons to boastfully tell me that they never do a blood count for an acute appendix, but they never make mention of all the normal appendices that they remove. A case that I saw recently can best illustrate the possibility of confusion from a blood count. Extreme fatigue prompted the patient to have a general examination. Badly infected tonsils was the chief

finding. Blood count was as follows: Hemoglobin, 62; leukocytes, 4,350. Differential count: Polymorphonuclears, 39; small lymphocytes, 61; large lymphocytes, 0.

All of the cells are old, mature cells, suggesting a chronic condition. This is a distinct tonsil count.

It happened that two days following the examination the patient suffered from what appeared to be an acute appendix clinically. Repeated count was: Leukocytes, 11,550. Differential count: Polymorphonuclears, 75; small lymphocytes, 20; large lymphocytes, 5.

Most of the above cells are very small and are new cells, suggesting an acute condition.

If one refers to any textbook, the last count is certainly not the classic picture, and yet when comparison is made to the count, which by mere fortune was taken only two days previous, it will be noticed that there is a decided leukocytosis with a marked increase in the polymorphonuclear cells, hence the count does show an acute appendix. In the appearance of the cells also, the first is definitely a chronic condition, while the second count shows as definitely an acute process.

My observations are based on some two thousand tonsillar bacteriological as well as hematological studies and on studies in many cases postoperatively. The clinical examinations were all made by someone else, and frequently in my presence, and I was otherwise acquainted with the full clinical picture. I also knew the treatment instituted and observed all results, hence am acquainted with all cases where tonsillectomy did and did not help and checked accordingly with the blood count.

In conclusion then, the culturing of tonsils and other bacteriological examinations are advocated. The search for the tubercle bacillus in tonsils is stressed, and the "tonsil blood count" is shown to be the surgeon's guide as to when diseased tonsils are the cause of systemic disturbances.

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